VOLUME 11
NUMBER 9
SEPTEMBER, 1958

ORAL SURGERY
ORAL MEDICINE
and ORAL PATHOLOGY

EDITORIAL

THE NEW DEPARTMENT OF STOMATOLOGY AT THE SCHOOL OF MEDICINE, BOSTON UNIVERSITY

Boston University has added to its well-known Medical School a Department of Stomatology for the postdoctoral education of dentists, especially those preparing for a specialty board certificate or intending to make a career of dental teaching and research.

Ten years ago this Journal was founded in order to provide an outlet for such clinical and research endeavors, the objectives of which were to "enlarge the horizon of dentistry."[1] Today this broad view of dentistry has been generally accepted. With the boundaries of our professional field thus expanded, it is impossible to include in an undergraduate dental curriculum all advanced knowledge, all the results of biologic and clinical research, and all the sciences basic to medical and dental practice which form a background needed to probe deeper into the dental unknown.

The need for graduate dental centers has become more and more evident. Some degree of advanced education has been offered by 73 per cent of the dental schools in the United States in the year 1956-1957, according to Conley[2] of the American College of Dentists. Conley also stated that the facilities for enrollment in most schools are quite limited. The American Dental Association furnishes a list of internships and residencies offered by the various institutions which are approved by the Council on Dental Education. The programs offered are difficult to evaluate. A great deal depends on the faculties. Often the instruction is provided by members of the undergraduate faculty who have neither the time to devote themselves to the graduate student nor the background required for teaching at the graduate level. In some of the existing programs, graduate students are utilized for teaching the undergraduate routine procedures, or they are left on their own initiative in large hospital clinics with little opportunity to work with and be carefully supervised by a qualified specialist. During the clinical years the graduate student benefits a great deal from association with competent and skillful half-time teachers whose major

connection with the institution is graduate-level teaching and who have no responsibility in an undergraduate curriculum. In oral surgery, such men generally arrange for the oral surgery student to assist at private operations. This exposure is an important additional benefit.

The postdoctoral program initiated at Boston University offers just such opportunities. The University has no undergraduate dental departments, and the entire staff will concentrate on graduate standards adapted especially for the accepted stomatologic specialties. Excellent clinical opportunities are offered at associated hospitals.

Other opportunities are offered in the departments of bacteriology, pathology, and biochemistry. These include the carrying-out of routine laboratory tests to get information regarding the general health of the patient and to undertake investigations of a scientific nature.

Personnel and facilities will be available for conducting research work, for it is believed by the staff that a proper attitude toward research and scientific investigation is an important part of graduate education.

With regard to the course in oral surgery which is being inaugurated this year, a certificate will be awarded after the first year of study, the basic year, to those fullfilling the requirements.

Graduate courses in the other recognized specialties will be offered next year, beginning in September, 1959. Refresher courses in these fields will be announced and will be offered during the coming academic year.

K. H. T.

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2. Conley, F. J.: Opportunities at the University or Dental School, J. Am. Coll. Dentists 25: 16, 1958.

OPERATIVE ORAL SURGERY

AMELOBLASTOMA

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THIS tumor is most frequently found in the molar region of the mandible. It may be solid or cystic, monocystic or polycystic. This tumor is considered an epithelioma of the basal-cell variety, not benign nor highly malignant. It may appear radiographically as a radicular or dentigerous cyst, although such cysts have clear outlines and the abutting teeth are pushed aside whereas in ameloblastoma the teeth may be eroded. It not completely removed at the first operation, the tumor may occur in a malignant form which requires radical surgery.

Ameloblastoma may cause a progressive facial disfigurement. The slowly growing swelling, rather than pain, brings the patient to the dentist; pain is only occasionally present. Soreness may develop as a result of trauma during mastication, and ulcers, fistulous tracts, and secondary infections are not uncommon.

The most striking feature of ameloblastoma is a definite resemblance to the enamel organ. The morphology of the tumor indicates that it is derived from the epithelium of the fetal enamel organ or the tissue forming it. In the histologic specimens it has been possible to demonstrate very clearly how the proliferations extend from the lining stratified epithelium to form the epithelial areas, which bear a striking resemblance to the enamel organ in the different stages of its development.

CASE 1

A 31-year-old man complained of enlargement of the right side of his face. Dental repair had been done on numerous occasions, but he had never had a dental x-ray examination.

Examination.—On the right side of the face, at the angle of the mandible, there was a smooth swelling. The oral mucosa appeared normal in color. X-ray examination revealed a cystic cavity within the body of the mandible. There was resorption of the roots of the molar teeth (Fig. 1, *A*).

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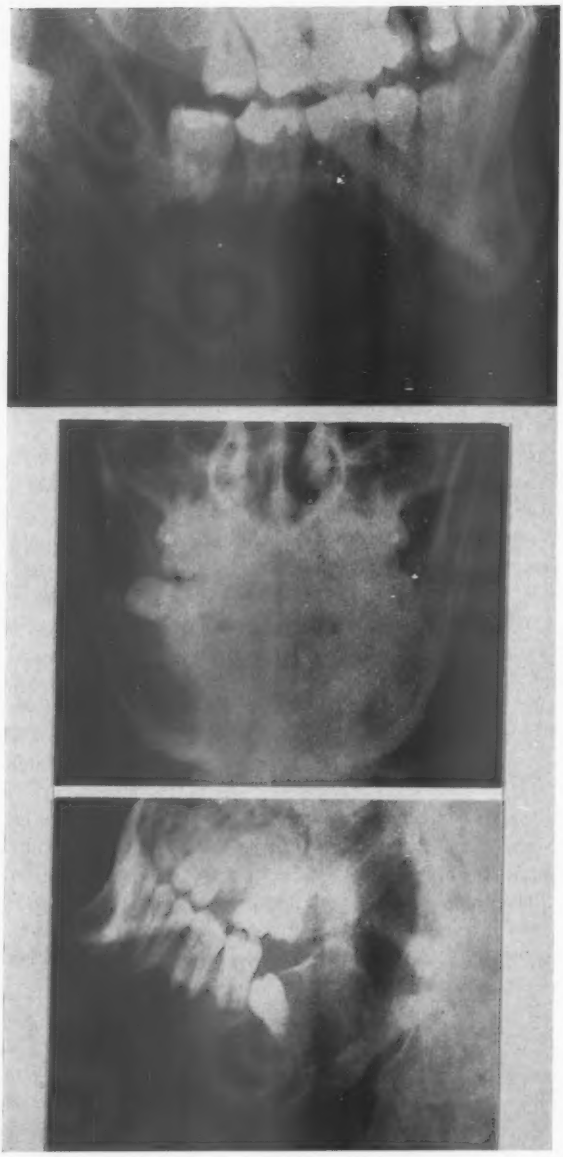

Fig. 1.—Ameloblastoma.

A, Case 1. Lateral radiograph demonstrates an ameloblastoma of the mandible. There is resorption of the roots, of the first, second, and third molar teeth.

B, Case 2. Postero-anterior radiograph reveals an ameloblastoma involving the right side of the mandible.

C, Case 2. Lateral radiograph demonstrates the polycystic lesion and an unerupted third molar.

Treatment.—Under local anesthesia, an incision was made into the mucobuccal fold at the first premolar, and the gum was separated at the gingiva labially around the second premolar and the first, second, and third molars. The exposed mandible appeared thin and elastic-like to touch. The bone was removed, first with a chisel and then with rongeurs, down to the inferior border of the mandible. With a curette, the tumor was freed along all margins and appeared solid. There was considerable bleeding. The second premolar, and the first, second, and third molars were then removed with a forceps. The area was made dry, and the lingual wall was found to be intact. The cavity was packed with small pieces of Gelfoam and closed with silk sutures.

Postoperative follow-up was uneventful.

The pathologic report indicated ameloblastoma.

CASE 2

A soldier, aged 21 years, was seen in a general hospital overseas with the complaint of "irritation in the third molar area."

Examination.—There was swelling intraorally in the retromolar area of the mandible on the right side.

X-ray Examination.—Cysts of the mandible were seen in the premolar—first molar area and also in the third molar region, extending to the condylar notch. The cystic cavities were separated by a bony septum (Fig. 1, *B* and *C*).

After the x-ray film was studied, the teeth were tested for vitality and, within the area, responded normally to both electric and thermal tests.

Procedure.—Following induction of general anesthesia, an incision was started in the cuspid area and carried posteriorly along the inferior border of the mandible. The mucosa was retracted, and perforations of the buccal plate were found in the first and third molar areas. The crown of the unerupted third molar was covered with a cystic membrane. Dense alveolar bone found below the apex of the second molar was removed with chisels. A cystic cavity was found, extending from the apex of the second premolar posteriorly to the condylar notch. With a curette, it was peeled out in one piece (including the third molar tooth). The roots of the second premolar and first molar extended into the cavity; the distal root of the second molar was reported. The three teeth were removed.

The cavity was washed with saline and then loosely packed with Gelfoam, and the mucosa was closed with 000 silk sutures. The pathologic diagnosis was ameloblastoma.

The patient remained in the hospital for six weeks and was then given temporary duty at the hospital.

In six months a partial denture was inserted, and the soldier returned to full duty.

CASE 3

A 46-year-old man was seen in the office in 1951 with the complaint of "swelling around the lower front teeth."

Examination.—The mucosa on the lingual and buccal surfaces about the mandibular incisor teeth appeared to be grossly hypertrophied. The left second incisor was loose.

Dental radiographs revealed several small cysts and a large cyst causing a divergence of the cuspid and second incisor roots. The root of the second incisor was resorbed.

Procedure.—The patient was admitted to Doctors Hospital. Following induction of general anesthesia, an incision was made along the gingiva from the second premolar on the left side to the second premolar on the right. The mucosa was retracted, and the bony surface was found to be perforated around the left second incisor. The alveolar bone was removed with rongeurs. One large cavity and several small ones were present. The tissue in the large cavity was fibrous. The anterior teeth were removed with a forceps, and the cystic and fibrous tissue was removed with a curette. After the cavities were made clean, the hypertrophied tissue was removed and the cavity was washed with saline. The cavities were packed lightly with Gelfoam, and the clean mucosal surfaces were closed with 000 silk sutures.

The pathologic report revealed ameloblastoma. The patient was discharged from the hospital on the third postoperative day, and the sutures were removed in one week. A partial denture was designed and inserted in three weeks' time.

Comment.—This patient, a local businessman, has been checked every six months for five years and has not had a recurrence.

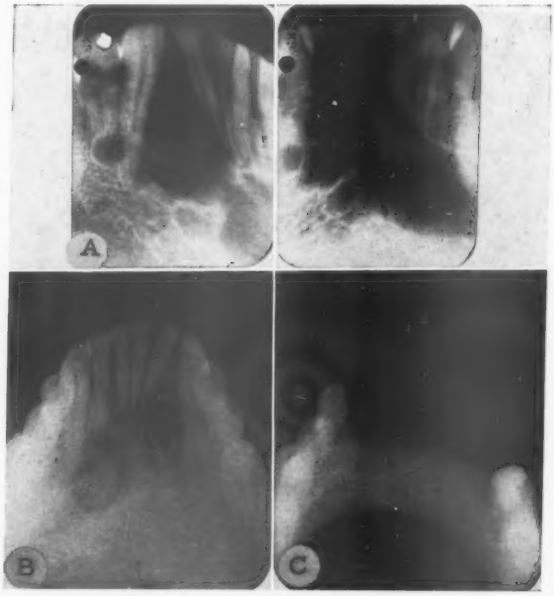

Fig. 2.—Case 3. *A*, Dental films reveal polycystic ameloblastoma at the symphysis. *B*, Occlusal film of same patient before surgical removal of tumor. *C*, Occlusal postoperative film made five years after surgery.

CASE 4

A female nurse, aged 36 years, was first seen in the office in 1949. She stated that "a lower wisdom tooth had been removed ten years previously" and she "felt a swelling back of the second lower molar."

Examination.—The mucosa posterior to the second premolar was elevated, and there was a smooth swelling on the buccal surface.

X-ray examination revealed a large cystic cavity extending from the mandibular first molar area on the right side to the condylar notch.

The first and second molars responded normally to vitality tests.

Procedure.—The patient was admitted to Jackson Memorial Hospital on July 6, 1949, and, under general anesthesia, an incision was made along the gingival margin from the second premolar and carried along the superior surface of the ascending ramus.

The mucosa was retracted, and bone of eggshell consistency was encountered. The cystic cavity was completely uncovered, and the mass was removed in one piece. It had a leathery consistency to touch. The roots of the molars were not seen and were not removed.

The cavity was cleansed with saline and packed loosely with Gelfoam. The mucosa was closed with silk sutures.

Fig. 3.—Case 4. *A*, Lateral radiograph made in 1949. *B*, Postoperative lateral radiograph made in 1951. *C*, Follow-up radiograph made in 1956 reveals filling-in of new bone. The pathologic diagnosis was ameloblastoma.

Convalescence was uneventful, and the patient was discharged on the fifth postoperative day.

Follow-up.—The patient is still on duty as a nurse and has been seen periodically for seven years. For six months she had "a numb feeling" on the operated side. There has been no recurrence of the tumor.

COMMENT

In general, oral surgeons today feel that if care is taken to remove the tumor completely, in the first operative procedure, recurrences do not occur.

It is true that there have been recurrences, but the older practitioners report that in their personal experience this has happened because remnants of the original tumor were left behind.

Correct diagnosis, careful planning, and meticulous surgical procedure are advised in the treatment of ameloblastoma.

POLYCYSTIC AMELOBLASTOMA OF THE MAXILLA

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AMELOBLASTOMAS of the jaws have been reported many times in the medical and dental literature. All authorities agree that these odontogenic tumors are found more often in the mandible than in the maxilla. It is with this thought in mind that I am reporting this case, which also is of interest because the more interesting lesion was primarily masked by an acute alveolar abscess in the area.

CASE REPORT

H. C., a 40-year-old white man, was referred to me by a local dentist for the removal of an acutely infected upper left maxillary cuspid (Fig. 1) and first premolar. The condition was associated with extensive facial cellulitis. Routine investigation into the physical condition of this patient revealed no contraindication to proceeding with the necessary extraction and drainage of the acute abscess. He therefore was given 15 c.c. of Pentothal sodium intravenously, and the procedure was carried out uneventfully. As the teeth were removed, 20 c.c. of foul pus was evacuated. The patient was given 600,000 units of penicillin daily for three days, which adequately controlled his acute infection. When this patient was seen again in my office ten days later, there was no sign of infection, and the patient felt well. It was noticed, however, that there was a definite asymmetrical outline of his face. On questioning the patient, it was apparent that he was unaware of this situation. It was his feeling that this was his normal appearance. Further roentgenograms of the region revealed an impacted second premolar embedded in bone which had a honeycomb or soap-bubble appearance (Fig. 2). At this time the embedded tooth was removed and a specimen of bone was submitted to the New Britain General Hospital for microscopic examination. The pathology department reported ameloblastoma (Fig. 3).

Hospital Course.—The patient was admitted to the New Britain General Hospital for more definitive surgery. Physical examination on admission revealed the patient to be well developed and apparently in no acute distress. His only complaint at that time was of slight swelling of his face on the left side. Heart and lung examinations were negative, and there was no history of any chronic systemic disease.

Laboratory Data.—A blood count disclosed the following: Hemoglobin 101 per cent, 15.7 grams; white blood cells 8,450 with 78 neutrophils, 1 band form, 1 eosinophil, 17 lymphocytes, and 3 monocytes. Urinalysis revealed slightly cloudy, dark yellow urine, acid in reaction, 0 albumin, 0 sugar, specific gravity qns., 1 plus bacteria LPF, 2-4 epithelial cells HPF, 2 plus mucus, 1 plus uric acid, 1 plus amorphous urates. The patient's blood was group O, Rh positive.

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X-ray Findings.—Films of the alveolar margins in the region of the premolar and adjacent molars showed a destructive process that measured approximately 1.5 by 2 cm. in diameter. X-ray films of the sinuses taken on the same day showed the right maxillary sinus to be clouded and extremely suggestive of destructive type of tumor (Fig. 4). There was rarefaction in the floor of the right maxillary antrum.

Fig. 1.—Cuspid and premolar responsible for infection and facial cellulitis.

Fig. 2.—Embedded premolar and honeycomb bone appearance.

Operation.—Under general anesthesia with nasoendotracheal intubation, following the usual intraoral and extraoral preparation, a classical incision was made over the crest of the alveolar ridge on the right side from the central incisor region to the third molar area. The mucoperiosteal flaps were turned back with considerable difficulty, since the tumor seemed to be adherent to the periosteum in both lateral and palatal aspects. However, the flaps were successfully detached. Then, with rongeurs, osteotomes, and a large antral curette, the entire floor and lateral wall of the maxillary antrum were removed in one large swoop. Fragments of the tumor could still be visualized, however, in the palatal region and along the medial wall

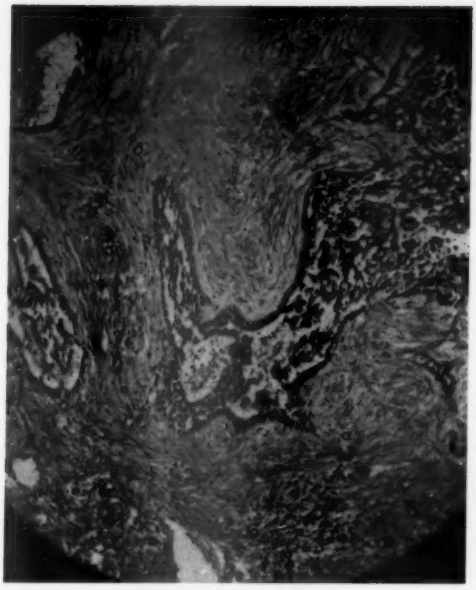

Fig. 3.—Biopsy specimen of lesion. Diagnosis: ameloblastoma.

Fig. 4.—Note cloudy appearance of right antrum.

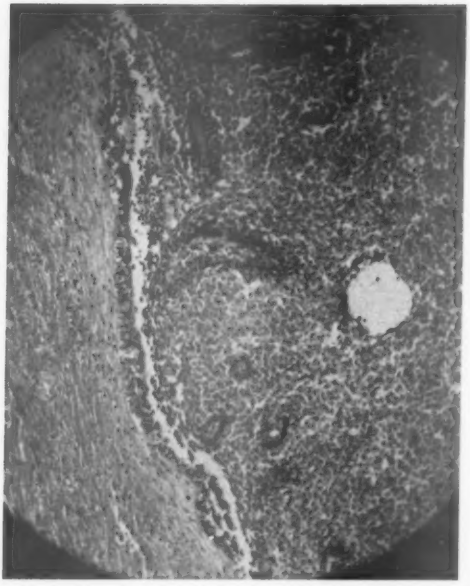

Fig. 5.—Surgical specimen (microscopic) showing tumor cells aligned in regular order and in single layer.

Fig. 6.—Note oral-antral fissure.

of the antrum. These areas were curetted until normal bone was approached. The entire wound looked exceedingly clean. Normal bone and tissue could be visualized in all aspects. Portions of the mucoperiosteal flaps were excised and submitted for microscopic analysis in order to ascertain whether the entire tumor had been circumvented. Following this, a nasal antral opening was made. This opening measured approximately 2 cm. in diameter. The antrum was then packed with a long strip of petrolatum gauze, and its ending was inserted through the naso-antral opening and allowed to hang free from the right nares. Three teeth had to be sacrificed during this procedure. The mucoperiosteal flaps were turned back and sutured into position with interrupted 000 silk. This effected a complete closure of the oral antral opening. The patient withstood the procedure well and left the operating room in good condition.

Microscopic examination of the surgical specimen revealed sections of the soft tissue fragments showing dense fibrous tissue containing multiple circumscribed zones of epithelial tumor, some solid and others cystic. The tumor cells were small and had nuclei of uniform size. Where they formed the lining of a cyst, the cells were aligned in regular order, usually in a single layer (Fig. 5). Where they formed solid or syncytial masses, no particular order was present and cytoplasm outlines of individual cells could not be distinguished. There were rare mitotic figures. A few small islands of tumor showed differentiation toward a squamous type of epithelium. A section of bone, prepared after decalcification, showed tumor like that described involving periosteum and marrow spaces. Sections of the two separate fragments of mucous membrane showed intact surface epithelium. No tumor was seen in the subepithelial connective tissue. Perivascular plasma-cell infiltrate and edema were present.

Pathological Diagnosis.—Ameloblastoma (maxilla); fragments of squamous mucosa showing chronic inflammation (from mucoperiosteal flaps).

Fig. 7.—Prosthetic appliance. (Courtesy of Dr. Sol Cohen, Kensington, Connecticut.)

Postoperative Roentgenograms.—Re-examination of the facial bones and paranasal sinuses revealed that a portion of the superior maxillary ridge on the right had been removed, including the lateral wall of the right antrum. Considerable mucosal thickening existed in both maxillary sinuses at this time, with somewhat less clouding of the right antrum than in the previous examination.

Postoperative Course.—Postoperatively, this patient did exceptionally well and experienced very little pain and swelling. Five days after the operation, the antral packing was

removed through the nares on the right side. During his convalescent period, the patient developed a reaction to penicillin and therefore was taken off penicillin and given Terramycin along with Pyribenzamine. He was discharged to my office for removal of sutures. It was apparent at this time that an oral-antral fistula was to occur (Fig. 6). Repeated attempts to close the fistula were unsuccessful, in spite of the fact that there was an adequate naso-antral opening. The patient was subsequently referred back to his dentist for construction of a dental prosthesis (Figs. 7 and 8).

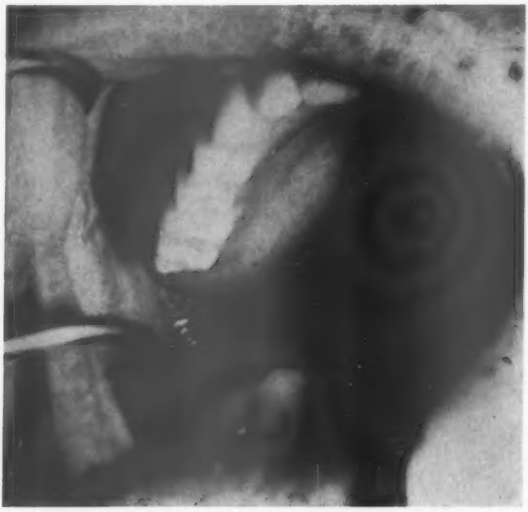

Fig. 8.—Appliance inserted into mouth of patient.

COMMENT

A complete discussion of these odontogenic tumors can be had by reading Dr. Kurt Thoma's textbook entitled *Oral Pathology*. It has been apparent to me that there are few reports in the literature demonstrating maxillary ameloblastomas.

MULTIPLE MYELOMA WITH MAXILLARY MYELOMATOUS EPULIS AND MALIGNANT PHEOCHROMOCYTOMA

Report of a Case

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THIS is a case report of an unusual combination of malignant diseases which clinically manifested itself first with a severe hematopoietic disorder caused by plasma-cell myeloma. Diagnosis of this neoplastic disease was based on corroborative laboratory findings, roentgenographic evidence, and biopsy evaluation. A second asymptomatic and unusual tumor of the adrenal medulla (malignant pheochromocytoma) was disclosed at necropsy.

The presence of an oral myelomatous epulis extruding from the maxilla was the first visible evidence of the disease process.

CASE REPORT

The patient, a 51-year-old white man, was admitted in a state of shock to a local hospital on Nov. 9, 1956. Twenty-four hours prior to admission he had passed a large tarry stool before leaving for work. During the evening, he complained of severe pain in the stomach, chest, and left arm. The next morning he had three fainting episodes, during which he appeared unconscious but was not cyanotic and had no seizure type movements. Vomiting was denied.

Examination.—The patient showed signs of clinical shock. Rectal temperature was 104° F. The skin was cold, very moist, and inelastic. He was markedly dehydrated. Three plus muscle guarding of epigastrium and upper right quadrant was present with tenderness in the epigastrium, at the xiphoid process and the lower sternum, and along the right fifth rib and intercostal space lateral to the midaxillary line. No masses were found in the abdomen. The liver, kidneys, and spleen were not palpable. Considerable tarry stool was found in the rectal ampulla. Barium swallow was negative for esophageal varices and gastric lesion.

The initial laboratory studies were as follows: *Hematology*—red blood cells, 1,976,000 per cubic millimeter; hemoglobin, 5 grams per 100 c.c.; hematocrit, 17 per cent. *Urinalysis*—4 plus albumin. Sinus tachycardia was the only electrocardiographic finding.

Treatment.—The patient received nine pints of whole blood in forty-eight hours. The blood elements were increased significantly: red blood cells, 4,520,000 per cubic millimeter; hemoglobin, 10 grams per 100 c.c.; hematocrit, 36 per cent. A sigmoidoscopy and barium enema were negative. The patient's condition improved remarkably, but he continued to

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Sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are a result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

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complain of pain over the xiphoid region and in the epigastrium. Also, pain in the right chest increased when he coughed, changed position, or breathed deeply. The blood platelet count was found to be decreasing sharply: 84,000 per cubic millimeter (November 13) to 64,000 per cubic millimeter (November 15). Initial clot retraction occurred at four hours, but retraction was incomplete after twenty-four hours. The prothrombin time was normal.

The decreasing platelet count prompted a bone marrow study. The latter indicated metastatic invasion of bone marrow. The most likely diagnosis suggested was myeloblastic leukemia or multiple myeloma. No megakaryocytes were seen in the marrow preparation.

Further Comprehensive Study of the Patient.—On Nov. 19, 1956, the patient was admitted to the medical service of the Veterans Administration Hospital for further diagnostic procedures and treatment. He was not in acute distress but complained of persistent tenderness in the lower end of the sternum and the chest wall at the right costal margin. A "scratchy" sound was heard throughout the cardiac cycle and was not related to respiration. The liver was enlarged to percussion, and the liver edge was palpable about two fingerbreadths below the right costal margin. It was moderately tender. Numerous soft pedunculated masses were scattered over the back and arms, and several café-au-lait pigmentations were found in the skin of the back.

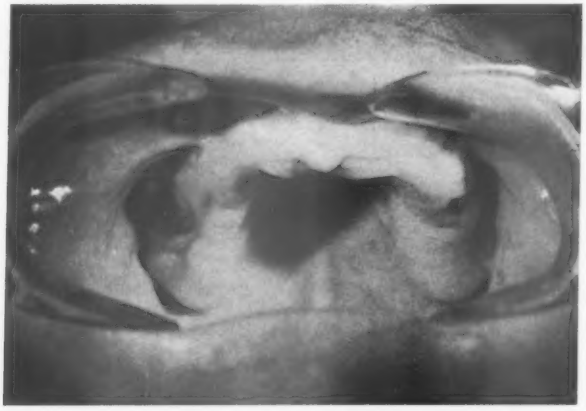

Fig. 1.—Photograph of myelomatous epulis of the right alveolar ridge. The palatal lesion is not visible in this view.

The patient was seen at a routine dental service oral examination. He was wearing a serviceable maxillary denture in occlusion with eight anterior mandibular teeth. In the upper right second molar area an ovoid, dark reddish blue tissue mass, 1 by 1 cm., was seen extruding from the gingiva. The lobulated surface was soft to touch, did not blanch or bleed under pressure, and was not painful. The lesion extended upward into the mucobuccal fold where indentation of the denture border could be seen coursing through the superior portion of the lesion. Inserting the denture caused a pedunculated lip of the tissue to hang over the lateral surface of the denture flange. Admitting that the denture did not fit as well as it had, the patient complained that lately his right upper jaw felt sore and that relief was gained by removing the denture for several hours. A slightly raised, flat, circular reddish blue lesion, 5 by 5 mm., was present in the posterior palatal mucosa near the midline of the right hard palate. It was nonpainful and was completely covered by the inserted denture. The patient did not know that these lesions existed in his mouth (Fig. 1).

Laboratory Examinations.—During November and December, the leukocyte count varied from 4,500 to 8,700 per cubic millimeter; hemoglobin, 13.5 to 8.8 grams per 100 c.c.;

and the platelet count, 140,000 to 70,000 per cubic millimeter. The prothrombin time was within normal limits at all times. Clot retraction required from thirty minutes to six hours for completion. Serum bilirubin was 0.88 mg. per cent. Blood urea nitrogen was 19.1 mg. per cent (Dec. 6, 1956). The total blood protein was 7.00 Gm. per cent to 6.92 Gm. per cent. Urinalysis revealed a 4 plus albumin in all specimens, but no Bence-Jones bodies were found.

Fig. 2.—Radiograph of skull with osteolytic lesions of the frontal bone. Lesions, outlined by arrows, are not as evident in photographic print as in original radiograph.

From January until the patient's death on March 18, 1957, the leukocyte count varied from 3,400 to 19,800 per cubic millimeter (March 3); hemoglobin, 7.2 to 10.25 grams per 100 c.c.; and platelet count, 66,000 to 180,000 per cubic millimeter. Clot retraction was delayed and required from one to six hours (average, four hours). Plasma cells ranged from 1 to 15 cells per count of 100 leukocytes. Nucleated red blood cells were seen frequently. The blood calcium varied from 4.38 to 4.94 mEq. per liter; sodium, 140 to 137 mEq. per liter; potassium, 4.34 to 4.26 mEq. per liter; and HCO_3, 17.0 to 10.8 mEq. per liter. Blood uric acid was reported to be 10.8 mg. per cent (March 13). The urea nitrogen progessively increased to 78.9 mg. per cent (March 15). The urine persistently contained 4 plus albumin.

Roentgenographic Data.—On Nov. 19, 1956, radiolucent focus was demonstrated in both frontal bones (Fig. 2). No other abnormalities were seen in the skull.

On Nov. 29, 1956, the cancellous alveolar process of the right maxilla appeared demineralized. The bony plate was absent. The margins of the focus were irregular and poorly defined, and the associated mucoperiosteum had a soft tissue projection adjacent to the osteolytic area (Fig. 3). No lesions were seen in the mandible.

On Dec. 17, 1956, examination of the thoracic spine and lumbosacral areas showed demineralization of the bones with cupping of the vertebral bodies and partial destruction of the articular surfaces of T_4 and T_5; a peculiar expanding lesion of the sacrum with erosion of the posterior margin of L_5; diffuse granular porosity of the spine and ribs; and a defect in the gastric cardia, which was interpreted as possibly due to pressure from the liver or a retroperitoneal mass.

Fig. 3.—Intraoral radiograph showing osteolytic lesion of maxillary alveolus in molar region. Shadow of soft tissue epulis is indicated by arrows.

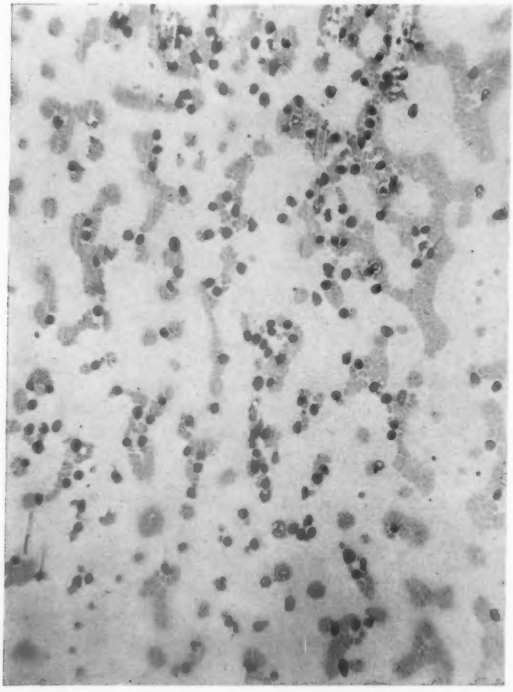

Fig. 4.—Photomicrograph of bone marrow smear. The excessive number of anaplastic reticuloendothelial cells is evident.

On Dec. 31, 1956, an essentially negative upper gastrointestinal tract was reported after a barium swallow.

On Jan. 2, 1957, a chest film demonstrated fracture of the left ninth rib in the axillary area and fractures of the fifth, sixth, and seventh ribs on the right with adjacent soft tissue masses, the largest measuring 2 by 3 cm. There was an osteolytic area of the left clavicle with an adjacent soft tissue mass.

On Jan. 7, 1957, the radiologist reported the presence of a left upper quadrant retroperitoneal mass displacing the stomach anteriorly and laterally and the left kidney slightly to the left.

Tissue Biopsy Examination.—A bone marrow study on Nov. 17, 1956, showed many abnormal, rather anaplastic cells of reticuloendothelial origin (Fig. 4) with many plasma cells, some binucleated. No bone marrow giant cells were seen. Impression: plasma-cell myeloma.

Fig. 5.—Photomicrograph of biopsy specimen from oral epulis. The ulceration of the overlying epithelium is seen at the right. Plasma-cell infiltration of the corium is generalized.

Biopsy of the lesion in the maxillary alveolar ridge on Dec. 7, 1956, revealed ulceration of the mucosa (Fig. 5) with invasion of the submucosa by crowded, compact sheets of plasma cells supported by a sparse, delicate fibrillar stroma. Some of the fibrous septa were hyalinized. There was clumping of the plasma cells around some blood vessels, giving a distinct "collar" effect. Impression: plasma-cell infiltration.

Protein Electrophoresis.—Electrophoretic examination of the blood serum on Dec. 4, 1956, by moving-boundary (Aminco) and zone (paper, Durrum) tests demonstrated an

abnormal component in the serum. This migrated with the electrophoretic mobility of a beta type of globulin. The other components of the serum were present in normal amounts (Fig. 6).

Tests on blood serum and urine on Dec. 11, 1956, revealed abnormal components which migrated with the electrophoretic mobility of a beta type of multiple myeloma protein. Blood serum drawn on Jan. 3, 1957, was tested as unextracted serum and as a diethyl ether extract. Both showed abnormal migrating components. There were a slight apparent increase in gamma globulin and a slight decrease in albumin as compared with the patterns of Dec. 4 and 11, 1956, respectively.

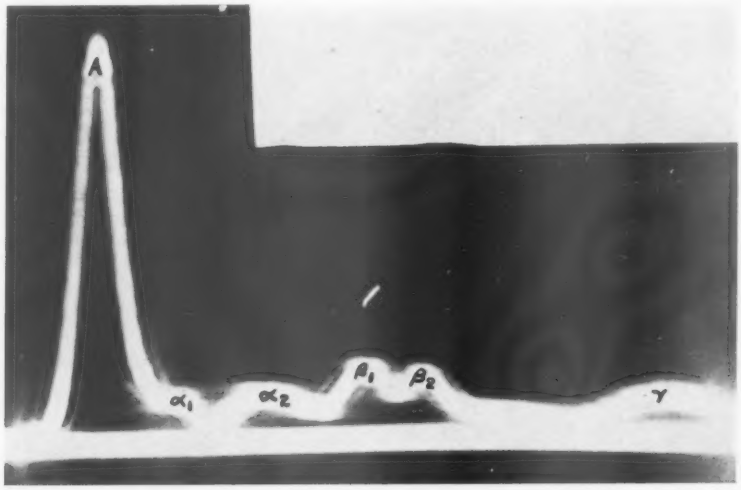

Fig. 6.—Electrophoretic pattern of the blood serum. *A*, Peak indicates that bulk of serum protein is albumin. α_1 and α_2, Alpha globulin. B_1 and B_2, Beta globulin. (B_2 peak represents migration of an abnormal protein component, compatible with multiple myeloma protein.) *r*, Gamma globulin.

Course.—Onset of the disease was characterized by acute hemorrhage from the gastrointestinal tract. Several blood transfusions were given and the patient's general condition improved remarkably. An abnormally low platelet count and a low blood clot retraction prompted a bone marrow study. Myeloma was suspected and the patient was transferred to the Veterans Administration Hospital for further diagnostic studies and treatment.

During December the patient complained principally of weakness and generalized chest and abdominal pain. Analgesics and small amounts of narcotics were sufficient to relieve discomfort. Urethane therapy started on Dec. 18, 1956, was continued to Jan. 9, 1957, and seemed to benefit the patient.

Several blood transfusions were given during January, with resultant improvement in the patient's condition. A cough developed, which produced considerable chest and abdominal pain.

In early February the patient was able to go home for several days. On his return, irradiation therapy to the lesion of the sacrum and to the maxilla was started. Total dosages of 2,050 roentgen units and 1,300 roentgen units were delivered to the respective sites. Urethane therapy was reordered.

Terminally, the patient required feeding by tube and intravenous solutions. Administration of narcotics daily was necessary to relieve the chest and abdominal pain. The cough was persistently productive, and breathing became progressively dyspneic. Later, there was darkening of the skin, unrelated to cyanosis. The patient died quietly on March 18, 1957.

Summary of Necropsy Changes.—Plasma-cell infiltration was present in the kidneys, spleen, liver, diaphragm, and the adventitia of the aorta. Both lungs contained leukemic infarcts, and the alveoli were filled with plasma cells and edema fluid.

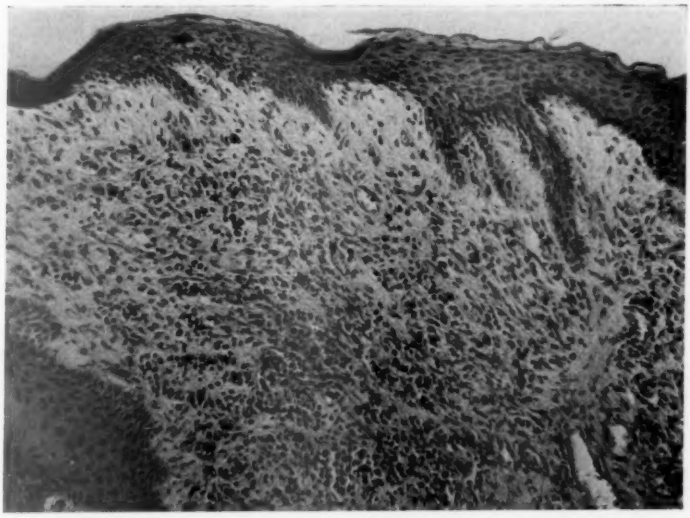

Fig. 7.—Photomicrograph of oral epulis tissue taken at necropsy. Following irradiation, surface epithelium is now intact and there is marked regression of plasma-cell infiltration. Some giant cells are present.

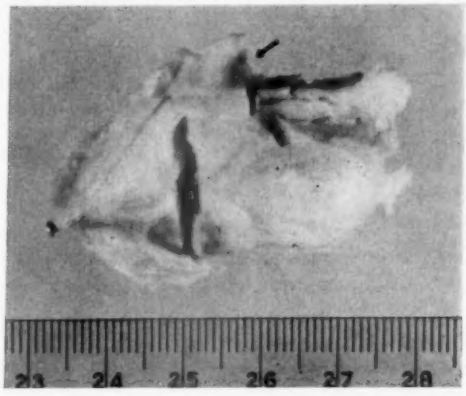

Fig. 8.—Gross specimen taken at necropsy of the maxillary alveolar ridge and portion of the palate. The heel of the maxillary tuberosity is at the right and the palate above. The epulis is grossly reduced in size after irradiation. The anterior portion of the palatal lesion is indicated by the arrow.

Osseous foci with replacement of much of the bone marrow were present in the vertebrae and sternum. The distal portion of the left clavicle was extensively infiltrated by plasma cells with expansion of the marrow cavity, thinning of the cortex, and invasion of the adjacent soft tissue. Multiple lytic foci were present in the ribs, and associated adjacent soft tissue invasion with pathologic fracture was common. Examination of sections of the

maxilla revealed postirradiation inflammation in an area devoid of trabecular bone. Plasma-cell infiltration still was present (Fig. 7) despite regression of the gross specimen of the myelomatous epulis (Fig. 8). Although the epithelium was intact over the palatal lesion, the mucous glands were hypertrophic and there was chronic inflammatory activity with plasma-cell infiltration deeper in the submucosa adjacent to the palatum durum (Fig. 9).

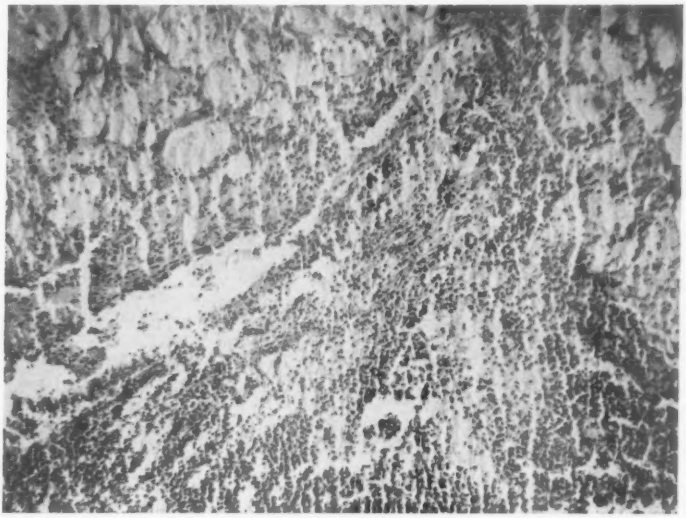

Fig. 9.—Photomicrograph of palatal lesion taken at necropsy. Plasma-cell infiltration is evident below the hypertrophic mucous glands.

Additional findings unrelated to the plasmacytoma included the following: malignant pheochromocytoma of the left adrenal gland with metastasis to the liver, both kidneys, and retroperitoneal lymph nodes; the jejunum contained leiomyomas; and the skin had multiple neurofibromas, one of which had metaplastic bone while another section included a lipoma.

Microscopic Diagnosis.—

1. Plasma-cell myeloma, extraskeletal and myelomatous.
2. Leukemic plasmocytic pneumonia with infaretion of lungs.
3. Malignant pheochromocytoma, left adrenal, with metastases to retroperitoneal lymph nodes, liver, and both kidneys.
4. Nephrosis, secondary to myeloma.
5. Leiomyoma in small intestines.
6. Neurofibromatosis of skin.
7. Mitral valvulitis.

Death was attributed to the extensive myelomatosis and the malignant pheochromocytoma with metastases.

CONCLUSIONS

This case presents an unusual combination of malignant and benign diseases.

A maxillary myelomatous epulis and palatal ulcer were the first visible manifestations of a fatal generalized myelomatosis. Plasma cells in the peripheral blood were found in addition to the extensive osseous foci and soft tissue lesions.

Diagnosis of the disease was established after summation of multiple procedures: tissue biopsy evaluation, marrow smears, roentgenographic examinations, electrophoretic identification of protein components, blood chemistry determinations, urinalysis, and blood cell counts.

An undiagnosed pheochromocytoma of the left adrenal gland was found at necropsy. Metastases to the liver, kidney, and retroperitoneal nodes had occurred. The radiologist's report of a left upper quadrant retroperitoneal mass was the only finding indicating existence of this asymptomatic malignant tumor.

Benign leiomyoma of the intestines was identified, and there were multiple neurofibromas of the skin.

The author wishes to express his thanks to those personnel of the Medical Service and the Laboratory Service whose major contributions made this case presentation possible.

PREGONIAL APPROACH TO THE SUBCONDYLAR AREA

*Jerome C. Stoopack, Captain (DC) USN**

CONSERVATIVE immobilization by means of closed reduction is adequate treatment in the majority of mandibular fractures of the condyle or subcondylar region. This conservatism is dependent upon the presence or absence of fracture displacement and the extent of such displacement. The simplest approach that results in anatomic alignment of the fragments, a normal healing process, and a functional mandible with good occlusion is the method of treatment to be preferred.

In the case to be reported there was marked overriding of the fragments, with the fractured end at an angle to the ramus and extending over it laterally. It was felt that conservative treatment would result in a poor occlusal relationship, with eventual strain and damage to the opposite normal joint. Therefore, open reduction with transosseous wiring was decided upon as the method of treatment. Since the fracture line appeared to be at the level of, or lower than, the mandibular notch, the pregonial approach was chosen to facilitate visualization of the fracture line and at the same time to avoid damage to the branches of the facial nerve.

CASE REPORT

Present Illness.—The patient, a 20-year-old white man, presented on April 5, 1954, with pain, swelling, and loss of function of the mandible. On April 4, 1954, he had been in a fistic altercation with an unknown assailant in Yamanaka, Japan. He was transported and admitted to this hospital for further evaluation and treatment.

Examination.—This revealed a well-developed, well-nourished white man in obvious distress. He was in great pain, but he was alert and well oriented. General examination was negative except for the local findings.

There was severe edema over the body and submandibular aspect of the right mandible. Some edema and tenderness were evident over the left condylar area. The oral cavity was bloody, although bleeding was minimal. Movement and crepitus could be demonstrated in the lower first molar area. Mandibular excursions were extremely painful and malocclusion was severe, although the teeth could be brought into contact posteriorly with deviation to the left. The maxilla was intact, and the patient had all of his teeth with the exception of several third molars. The rest of the examination was negative.

The opinions and conclusions contained herein are those of the writer and do not necessarily reflect the official views of the Department of the Navy or of the Department of Defense.

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Laboratory Findings.—Except for a total white blood cell count of 10,000, routine blood studies, bleeding time, clotting time, and serology were within normal limits. Urinalysis was unremarkable.

Radiographic Findings.—Routine chest views were negative. The mandibular and temporomandibular series revealed a fracture in the left subcondylar region, with lateral displacement and overlapping. The head of the condyle was medial, slightly anterior, and inferior to its normal position in the glenoid fossa (Fig. 1). There was also a compound fracture of the right mandibular body with distraction in the first molar area; this molar was within the fracture line.

Treatment and Course.—Antibiotic therapy consisting of Terramycin, 500 mg. every four hours in chocolate milk, was started along with a high-calory vitamin and protein liquid diet. Sedation was ordered; this consisted of Demerol, 75 mg. intramuscularly every four hours if necessary for pain, and Nembutal, 100 mg. intramuscularly if necessary for sleep.

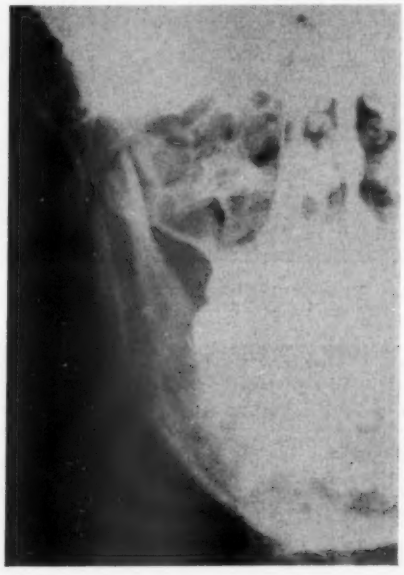

Fig. 1.—Posteroanterior radiograph of left subcondylar area. This and the following radiograph were taken subsequent to an attempt at conservative closed reduction by means of arch bars and intermaxillary wiring. It is seen that no appreciable improvement was effected by this procedure, as the admission radiographs disclosed approximately the same amount of displacement. These radiographs were taken on April, 9, 1954.

On April 6, 1954, under Xylocaine block and infiltration anesthesia, the lower right first molar, which as stated previously was in the fracture line, was extracted. Following this, maxillary and mandibular arch bars were wired into position (Fig. 2). With the teeth in anatomic occlusion, the jaws were then immobilized by means of intermaxillary wires.

Radiographs taken when the patient was more comfortable revealed that there was no change in the position of the fragments at either fracture line following this conservative procedure. Therefore, open reduction was scheduled.

Operation.—On April 11, 1954, following Nembutal, morphine sulfate, and scopolamine premedication and under Pentothal sodium and nitrous oxide-oxygen nasal intubation

anesthesia, the fracture was reduced and immobilized by open reduction. The intermaxillary wire traction was removed prior to intubation. The area to be operated upon was prepared in the usual manner. A pregonial incision was made 1 cm. below the inferior border of the mandible. It was carried posteriorly and parallel to the inferior border from the first molar area, following the angle, and then obliquely upward to a point below the ear lobe. The tissues were carefully separated in layers by blunt and sharp dissection, exposing the fracture site, the angle, and a portion of the lower border. Bleeders were tied as necessary.

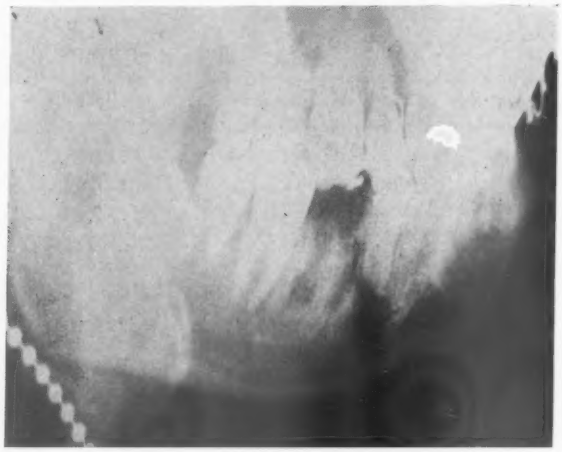

Fig. 2.—Left lateral oblique view of the mandible also taken on April 9, 1954. Distraction of the fragments can be seen.

Fig. 3.—Appearance of the incision area two days postoperatively. There was still some edema at this time, although the patient was quite comfortable.

Holes were then drilled in the proximal and distal fragments angled toward the fracture line. An accessory hole was drilled at the angle, and a wire was passed through for additional traction during reduction. An 0.014 inch stainless steel wire was then passed through the holes, the fracture was reduced into anatomic alignment, and the ends of the

wires were twisted securely and bent. The accessory wire was removed, and the incision was closed in layers by means of 00 chromic and 0000 Dermalon for the skin (Fig. 3). A pressure bandage was then applied. Elastic traction was placed intraorally when the

Fig. 4.—Appearance of the incision area seven weeks postoperatively.

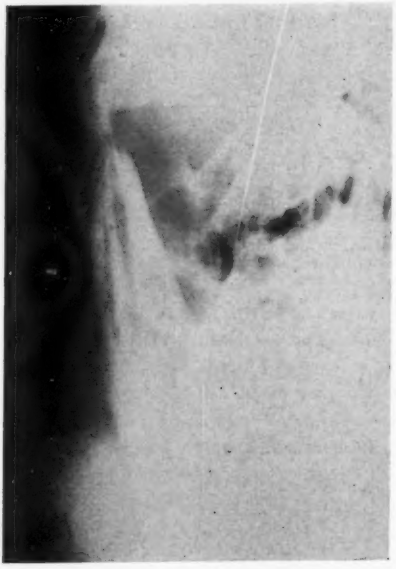

Fig. 5.—Postoperative Towne's view showing the same thing.

patient had awakened sufficiently and could understand the spoken word. On the following morning the elastic traction was replaced by intermaxillary wires, and the jaws were again immobilized with the teeth in anatomic occlusion.

The Terramycin was continued, and procaine penicillin, 300,000 units twice a day was started and continued for six days. Postoperative sedation consisting of Demerol, 100 mg. every four hours as necessary for pain, and Nembutal, 100 mg. if necessary for sleep, was ordered. The patient was kept on bed rest for two days and then was allowed to be up progressively more each day. By the fifth day he was fully ambulatory and comfortable. The pressure bandage was removed on the sixth day at which time all medications were discontinued. He was kept on the liquid diet, however.

Results.—Except for some postoperative edema which regressed by the eighth day, progress was uneventful. Every other suture was removed on the seventh day, and the remainder were removed on the ninth day. There was no apparent weakness of the branches of the facial nerve (Fig. 4).

Postoperative radiographs revealed both fractures to be reduced into anatomic alignment (Figs. 5 and 6).

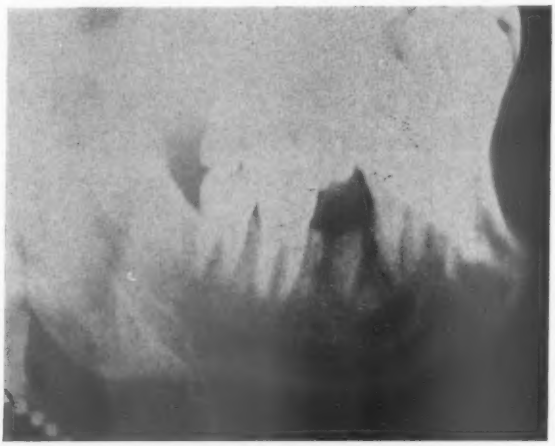

Fig. 6.—Left lateral oblique view of the left mandible taken on April 15, 1954. The distraction had been eliminated, and the fragments were in anatomic alignment.

The intermaxillary wires and arch bars were removed after six weeks, at which time there was slight trismus. This was relieved in four days by normal mandibular exercise.

Temporomandibular radiographs taken at this time revealed movement of both condylar heads during mandibular excursions, although there was some limitation.

All carious teeth were restored, prophylaxis was completed, and the patient returned to full duty on May 15, 1954, at which time a regular diet was tolerated with no pain or discomfort.

CONCLUSIONS

A case of displaced overlapped fracture of the mandibular condyle and distracted compound fracture of the opposite mandibular body, treated by open reduction and transosseous wiring via the pregonial approach, has been presented.

Excellent visualization of the fracture line is obtained by this approach in displaced condylar fractures at the level of or below the mandibular notch.

Damage to the branches of the facial nerve is minimal or absent following this procedure. Extreme care must be taken to avoid the inferior or cervical mandibular branch.

In the majority of cases, mandibular fractures, including fractures of the condyles, can be reduced conservatively by closed reduction. The simplest method of treatment that results in anatomic alignment of the fragments, a normal healing process, and a functional mandible with functional occlusion is the method to be preferred.

EXODONTIA

HYPOFIBRINOGENOPENIA

Sydney A. Rose, D.D.S., Hempstead, N.Y.

FIBRINOGENOPENIA may be congenital or acquired. The acquired condition is usually secondary to defective formation of fibrinogen, increase in fibrinogen consumption following intravascular clotting, or destruction or digestion of fibrinogen by fibrinolytic or proteolytic enzymes circulating in the blood stream.[1]

These forces destroying fibrinogen may be observed following severe trauma, shock, burns, hemorrhage, and surgical procedures. The mild activation of fibrinolysin which may be seen in most patients undergoing surgery is probably caused by the discharge of epinephrine. Bleeding seen secondary to intravascular proteolysis may assume a dangerous degree. If there is an acute fibrinolysis, severe hemorrhage will occur from all surgical wounds and into the skin and mucous membranes. Any sudden increase in bleeding during a surgical procedure is a warning of possible fibrinolysis and calls for immediate action with regard to diagnostic and therapeutic procedures. "Such minor surgical procedures as dental extractions may be followed by interminable, uncontrollable hemorrhage."[1] "The bleeding in cases of acquired hypofibrinogenopenia is often of extreme gravity and requires the prompt administration of fibrinogen (2-4 grams or more), as well as whole fresh blood."[2] "A wide variety of operations are included which would seem to indicate that hypofibrinogenopenia can occur in almost any operative procedure. No responsibility to type of anesthesia can be established since the proportion of cases receiving a particular drug corresponds roughly to that used in surgical patients throughout the hospital."[3]

Treatment of hypofibrinogenopenia should be limited to control of the immediate hemorrhagic crisis. The emergent treatment should be administered only if the bleeding occurs and not as a preventive measure. Patients with a low plasma fibrinogen level may go for long periods of time without hemorrhage. Once the hemorrhagic crisis is controlled, it usually does not recur as it may

in other bleeding diatheses. This procedure should be followed, for indiscriminate use of fibrinogen may bring about immunization to the protein in which the patient is deficient and thus greatly decrease the efficiency of applied therapeutics at some later crisis.

The indicated therapy at the time of evaluated crisis should include: (1) immediate transfusion of whole fresh blood, that is, blood under 24 hours old, as the fibrinogen does not remain active in older blood, and (2) intravenous administration of human fibrinogen, such as Parenogen (Cutter Laboratories, Berkeley, California), which contains 1 gram dried human fibrinogen per unit.

The patient may require as much as 6 grams of fibrinogen to control the hypofibrinogenopenia. The amount required can be determined by clinical observation, chemical determination, coagulation time, and observation blood clot.

CASE REPORT

A 61-year-old white woman was referred to my office for multiple extractions of badly carious and periodontally involved teeth.

The patient presented with a history of rheumatic fever, subacute bacterial endocarditis, acute bacterial endocarditis, and recurrent valvulitis. She had had many episodes of failure and had been digitalized for many years.

The patient was admitted to the Hempstead General Hospital on Dec. 4, 1956. Urinary and hematologic laboratory findings were normal. The physical examination revealed low blood pressure and tachycardia.

A cardiologic consultation revealed rheumatic heart disease, thyrotoxicosis, an enlarged heart, and auricular fibrillation.

Prior to surgery, the patient was put on prophylactic penicillin, 300,000 units intramuscularly every six hours, to be continued postoperatively for three days. The patient was cleared for surgery.

On Dec. 6, 1956, she was taken to the operating room. Following proper preparation and draping, the removal of the indicated teeth was started under endotracheal anesthesia.

During the procedure it was noticed that there was more than a usual amount of bleeding. Consultation with the anesthesiologist revealed that the blood pressure was remaining normal (140/80), with normal pulse. The patient had required only 0.5 gram of Pentothal sodium for induction and leveling and was being carried on nitrous oxide-oxygen 50 per cent. Local anesthetic was used regionally (2 per cent Monocaine and epinephrine) to obtain local anesthesia and hemostasis.

Blood was taken at this time and sent to the laboratory for examination, and the procedure was continued. Multiple sutures were placed and this seemed to aid in obtaining hemostasis. However, it was now noticed that, although the bleeding into the oral cavity was somewhat contained, the blood was welling in the tissues.

The laboratory report at this time showed a normal bleeding time of three minutes and fifteen seconds (Lee-White). The prothrombin time was as follows: patient, 18 seconds; control, 15 seconds.

The surgical procedure was now completed and the patient was taken to the recovery room. She was given whole fresh blood following cross-matching and typing.

Since the critical laboratory tests had proved within normal limits, they were ordered repeated and a fibrinogen level was also requested.

The fibrinogen level was reported as follows: positive at 1:100, partly defined at 1:50, and well-defined coagulation at 1:10 concentration. With this information, 50 c.c. of fibrinogen (containing 1 gram of fibrinogen) was prepared and administered.

The patient at this time presented with pale color. Her pulse was slow with fair quality. There was profuse bleeding of the oral tissues. Transfusion of the first 500 c.c. of blood was completed and a second was started at 12:50 P.M. The blood pressure was still acceptable at 140/80. At 1 P.M. the blood pressure was 130/80, and the bleeding was coming under control. At completion of the administration of the fibrinogen, the hemorrhage had stopped and what could be considered as normal oozing was observed.

At 4:15 P.M. the patient was returned from the recovery room to her room. The bleeding had come completely under control, and the blood pressure was 140/90.

The patient was discharged on Dec. 9, 1956. There were no further hemorrhagic episodes. She has been followed by her physician and has demonstrated fibrinogen levels within normal limits. I have seen her on periodic postoperative visits. She is now wearing dentures and leading a physiologically normal life.

CONCLUSIONS

Among the many bleeding diathases that confront the oral surgeon, hypofibrinogenopenia is one of extreme degree. Hypofibrinogenopenia has now emerged from the obscurity of isolated case reports as an established syndrome.[4] It presents a grave problem, for it may occur in the face of normal preoperative laboratory findings. The etiology may well be the surgical trauma concurrent with multiple extractions. The diagnosis must be expedient and the treatment heroic. It would be well for the oral surgeon to be sure of the presence of fibrinogen in the hospital pharmacy before assuming the responsibility of any oral surgical procedure.

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PERIODONTIA

THE POSITION OF THE MARGIN OF THE GINGIVA

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THE pocket, a prominent feature of periodontal inflammatory disease, is one of many links in the complex chain of events leading toward total destruction of tooth support. It grows by the constant collection of food and bacteria in its warm, moist, and secluded recesses. These organic aggregations calcify, further irritate the tissue, and hasten the deepening spread of pathology. To stop this self-perpetuating process, modern therapy is concerned not only with the removal of calcified irritants but with the elimination of the pocket as well.

Pockets are formed by coronal growth of the free gingiva, apical retreat of the epithelial attachment, or a combination of the two. A better understanding of these two factors would certainly help the clinician, both in preventing formation of the pocket and in eliminating it once it had formed. Toward these ends, this article will attempt to clarify and correlate the factors affecting the first of these processes, the changing position of the margin of the gingiva.

Any discussion of this subject should be based on Hirschfeld's classic observations[9, 10] of clinical and skull material. Hirschfeld noticed that a lingually malposed tooth possessed a thicker and more coronal alveolar buccal plate than was present against its properly positioned neighbors. The lingual plate, on the other hand, was thinner and ended more apically than on the adjacent teeth. Furthermore, the buccal (or lingual) alveolar crest was flat if the contiguous tooth surface was flat, or it curved coronally toward the proximal areas if the tooth surface was convex mesiodistally. Gingival shapes generally followed the same pattern, although not always to the same degree. These descriptions of bone and gingivae with normal crevice depths apply as well (with some important modifications) to the position of gingivae with pockets.

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PROMINENCE OR ACCESSIBILITY OF TOOTH SURFACE

The foregoing data suggest that tooth surface prominence is the most basic determinant in fixing gingival position. A workable empirical rule may be phrased as follows: *The position of the gingival margin is partly determined by the buccal (or lingual) prominence of the tooth surface adjacent to it, being more apical with greater prominence and more coronal with lesser prominence.*

Variations in Accessibility.—The degree of prominence (which is also a measure of accessibility to the action of muscles, saliva, and toothbrush) thus may be influenced not only by tooth position (Fig. 2) but by tooth shape and bone shape (Fig. 1) as well.

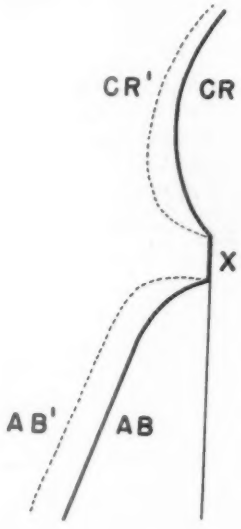


Fig. 1.—A point (*X*) on a tooth may be made more inaccessible by an exaggeration of the crown contour (*CR*) to *CR'*. The same effect is heightened when the alveolar bone (*AB*) becomes thickened (*AB'*).

A tooth surface is made less accessible when it is recessed by the exaggerated contours of the buccal (or lingual) aspects of crown and bone (Fig. 1).

Accessibility or prominence changes more rapidly as one travels a given distance mesiodistally on a tooth surface with a marked curvature as compared with a flatter tooth shape (Fig. 2).

The greatest combination of these influences and the most marked effects on gingival form exist at the interproximal area. Cramping of the gingival embrasure by either lowering the contact point or narrowing its mesiodistal

diameter will decrease the accessibility of the proximal surfaces and cause papillary overgrowth (Fig. 3). Similar effects are produced by encroachment on the buccal (or lingual) embrasures by crown contours (Fig. 4).

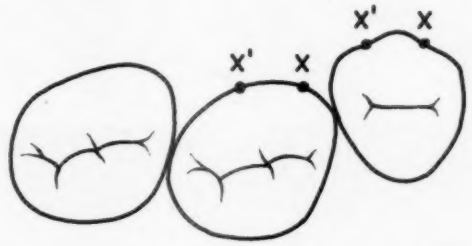


Fig. 2.

Fig. 3.

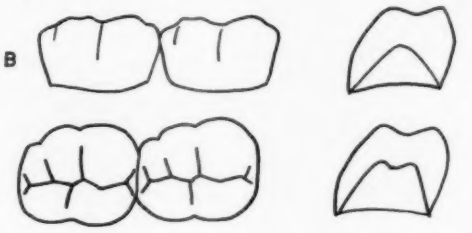


Fig. 4.

Fig. 2.—The point *X* on the center tooth is more inaccessible than the corresponding point on its neighbor because of the tooth's lingual malposition. *X* and *X'* are equidistant points on both teeth, but differences in buccal curvature produce greater changes in accessibility on the bicuspid than on the molar.

Fig. 3.—*B* illustrates a well-shaped triangular gingival embrasure and the pyramidal easily maintained papilla contained within. Encroachment on the embrasure dimensions (*A*) produces buccal and lingual papillary swellings on either side of the contact point. The resultant concave shape retains debris and is susceptible to inflammatory changes.

Fig. 4.—Results similar to *A* in Fig. 3 are also produced by coronal invasion of the buccal and lingual embrasures.

Effects on Gingival Position.—The correlation between accessibility and gingival position may be seen in any mouth or study model. The clinical photographs that follow were selected to emphasize the more important aspects of this relationship.

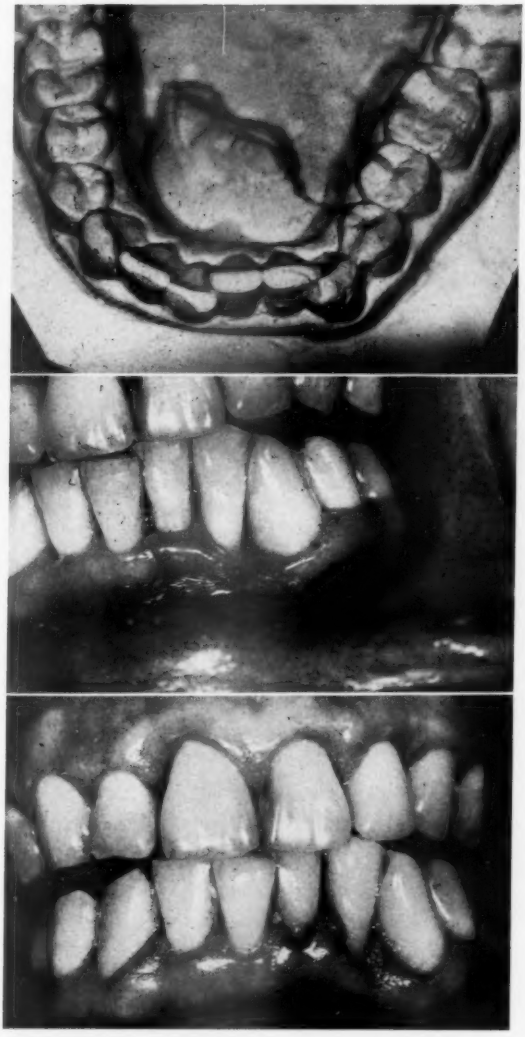
The margin of the gingiva in Fig. 5, *A* is thicker and more coronal at the lingually malposed lateral incisor than at the central incisor or cuspid. Labial orthodontic movement (Fig. 5, *B*) not only increased the accessibility of the labial surface of the lateral incisor, but it also caused a recession and tapering of the gingivae. In effect, orthodontics accomplished a gingivoplasty without benefit of surgery.

A.

B.

Fig. 5.—*A*, The gingival margin on the lingually malposed lateral incisor is more coronal than on the neighboring teeth. In addition, this margin is thick and inflamed.
B, Orthodontic (not periodontal) therapy has increased the accessibility of the lateral incisor and has produced a recession and a tapered healthy gingiva.

Since the bone labial to a lingually malposed tooth is thicker than adjacent labial bone (Fig. 6, *A*), it might seem that bone tapering alone would lower the gingival margin. Often the results of such therapy (Fig. 6, *B*) are temporary and end in regrowth of the free gingivae (Fig. 6, *C*). Bone tapering in this case could not overcome the degree of inaccessibility maintained by the relative positions of the teeth. Contrasted with the previous case, this was surgery without benefit.



A.

B.

C.

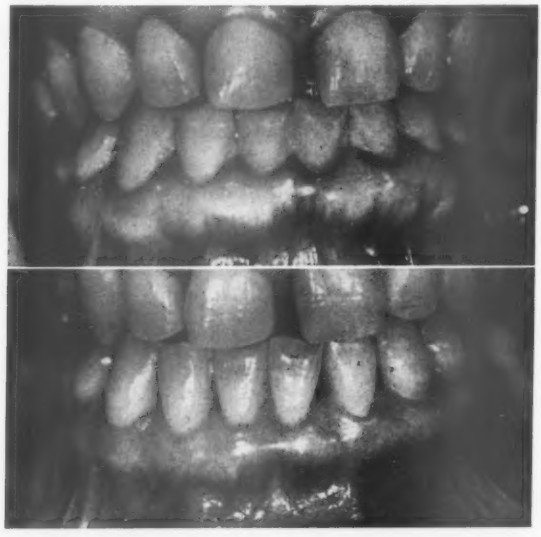
Fig. 6.—*A*, Malposition of $\overline{|\ 12}$ has resulted in a thick labial alveolar plate.

B, Gingival and osseous tapering has reduced pocket depth at $|\ \overline{123}$.

C, Regrowth of tissue within a year. The inaccessibility of this area was maintained by the relative tooth positions despite the gingival and osseous plastic reshaping.

Thickness of bone without tooth malposition produces inaccessibility with thicker, higher gingivae (Fig. 7, *A*) which is correctible by bone tapering (Fig. 7, *B*).

A.



B.

Fig. 7.—*A*, The inaccessibility fostered by thick bone, without tooth malposition, has resulted in thick, inflamed, slightly overgrown gingivae.
B, Postoperative view. Without malposition, bone tapering alone increased accessibility sufficiently to produce easily maintainable gingival forms.

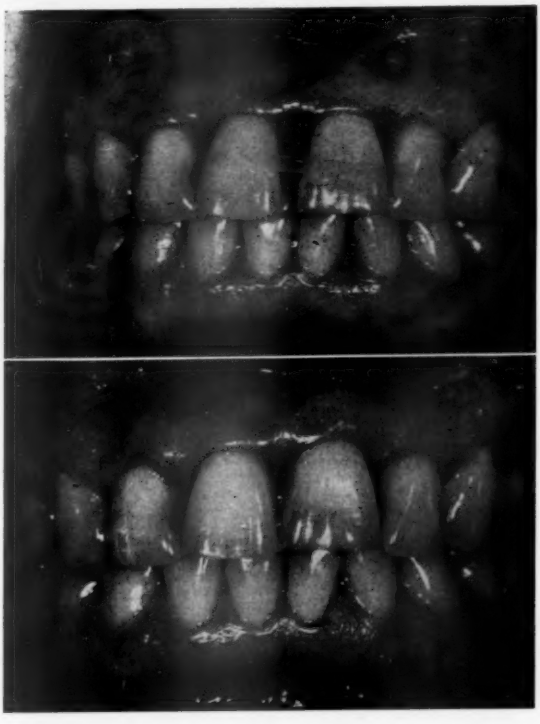
Fig. 8.—The papilla gradually lowers and flattens as the contact opens until an edentulous ridge appears.

Changes in accessibility due to degrees of mesiodistal curvature of labial surfaces (Fig. 2) are also reflected in changes of gingival position. This is

best illustrated by comparing the cuspid, which possesses the greatest mesiodistal curvature and the sharpest changes in gingival level, with the flat gingival contour on molars, which commonly display a fairly flat buccal surface.

The greatest inaccessibility exists at the interproximal area where, as one might expect, the gingiva rises to its summit. That the papilla is a structure born of tooth relationships is shown by its reactions to positional changes. As a tight contact opens slightly it permits a meager papillary

A.



B.

Fig. 9.—*A*, Flat papilla with open contact. *B*, Reappearance of papilla after contact was orthodontically closed.

growth without appreciably adding accessibility, but as the opening widens this factor gets the upper hand and the papilla gradually flattens (Fig. 8) until an edentulous ridge results. This is also a reflection of similar changes in bone contour going on at the same time.[16] Conversely, a papilla reappears as separated teeth (Fig. 9, *A*) are orthodontically moved together (Fig. 9, *B*).

The conditions for papillary formation are duplicated when natural or surgical recessions reach the level of a bifurcation (Fig. 10) where the root surface swings into the less approachable interradicular area.

Violations of the embrasures (Fig. 3, *A*) may be brought about by restorative dentistry or crowding of teeth. Crowns improperly imposed on a healthy area (Fig. 11, *A*) are the most typical examples of this effect (Fig. 11, *B*). They produce enlarged contact points, gold proximal surfaces that are thicker than the tooth substance they replace, and (particularly in the case of acrylic veneer crowns) overcontoured buccal surfaces (Fig. 1, *CR'*). Poorly contoured crowns have actually been used for "curing" recessions[12] by stimulating gingival growth and, incidentally, forming pockets!

Overshaped pontic-crown connections operate in the same manner. "Sanitary" ponties, on the other hand, preserve the accessibility of the proximal surfaces and leave the flat edentulous ridge contours unaffected.

Fig. 10.—Formation of a papilla where the interradicular space simulates the shape of an embrasure.

Crowding of teeth may create entirely different results when the gingival embrasure and interseptal bone are almost completely obliterated by extremely close tooth surfaces. In these cases (Fig. 12) the papilla is small or completely absent.[6] It is possible that pinching of the buccolingual center of the papilla divides it into labial and lingual segments, neither of which affects the height of the other. The height of the gingiva would then be influenced only by the easily accessible buccal (or lingual) tooth surfaces as they meet.

A photograph (Fig. 13) published by Dr. Wade[23] of London, England, highlights many of the points made above. This case, with the fairly common pubescent tendency toward gingival overgrowth, was subjected to three gingivectomies in the same area within one year. Swollen papillae repeatedly regrew where embrasures were cramped by malpositions. An ideal papilla remained only where a perfect embrasure was formed by the maxillary central incisors.

Since tooth shape, tooth position, tooth relationships, and bone shape may vary at different horizontal planes, the rise and fall of the gingival margin will tend to reflect the anatomic features characteristic of the particular level

A.

B.

C.

Fig. 11.—*A*, Well-formed healthy papilla between 4| and 3|.

B, Overgrowth of papilla and concomitant inflammation at 43| following invasion of embrasure by acrylic veneer crowns.

C, Re-establishment of papillary form and health. The embrasure was enlarged by surgically lengthening the occlusocervical dimension. This was accomplished by tapering and reducing the height of the interproximal bone.

Fig. 12.—The papilla is smallest between the mandibular central incisors where the embrasure is not merely diminished but almost completely obliterated by the combined effects of tooth anatomy and position.

Fig. 13.—Subjected to three gingivectomies within one year, a perfect papilla remains only where a well-proportioned embrasure exists, between the maxillary central incisors. Rounded papillae reappeared where tooth malalignments cramped embrasures. (From Wade, A. B.: *J. Periodont.* 25: 189-198, 1954.)

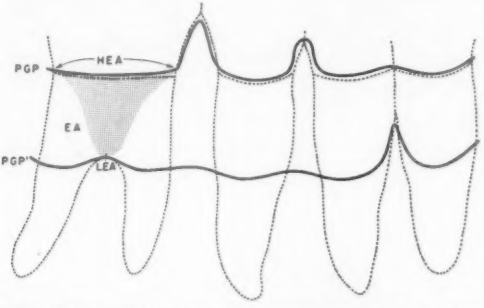


Fig. 14.—*PGP*, Physiologic gingival pattern, a rise and fall of gingival height governed mainly by changes in root surface prominence or accessibility.

PGP', Physiologic gingival pattern at a more apical level.

Shaded area represents a buccal pocket.

EA, Epithelial attachment bordering pocket.

HEA, High epithelial attachments at either side of pocket.

LEA, Lowest epithelial attachment of pocket.

Papillae form wherever tooth relationships provide the necessary inaccessibility, whether the gingivae are at a normal level (*PGP*) or exhibit a recession (*PGP'*).

LEA is used as a starting point to establish *PGP'* in order to eliminate the buccal pocket. The extension of the incision beyond the buccal surface of the molar depends on the presence or absence of adjacent pockets, tooth relationships, and the amount of remaining alveolar support.

If the pocket wall were simply excised, *HEA* would determine the future gingival pattern. As a result, the gingivae would grow to the original position and the pocket would return.

(Fig. 14, *PGP* and *PGP'*). Regardless of the initial point of reference, when this circumferential pattern of varying gingival heights is harmonious with accessibility gradients, it may be considered as a *physiologic gingival pattern.* Such a pattern cannot be defined in terms of stereotyped gingival shapes.

It should not be confused with Goldman's[5] *physiologic gingival contour,* which is a vertical cross-sectional profile of a gingival form that tapers as the margin is approached. His valid concept should be modified, however, when the presence of an "ideal" papilla implies and prolongs the coexistence of an adjacent pocket.

Mechanism of Accessibility and Gingival Position.—The mechanism of these gingival patterns is open to conjecture, but it is not unlikely that variations in tooth accessibility influence the effectiveness with which tongue, cheek, and lips mold the free gingivae. Thus, the papilla grows to a height and thickness at which molding action inhibits further expansion.

MODIFYING INFLUENCES

The basic relationships just discussed may be modified by a number of factors, such as gingival pathology, the pattern of epithelial attachment, and artificial abrasions.

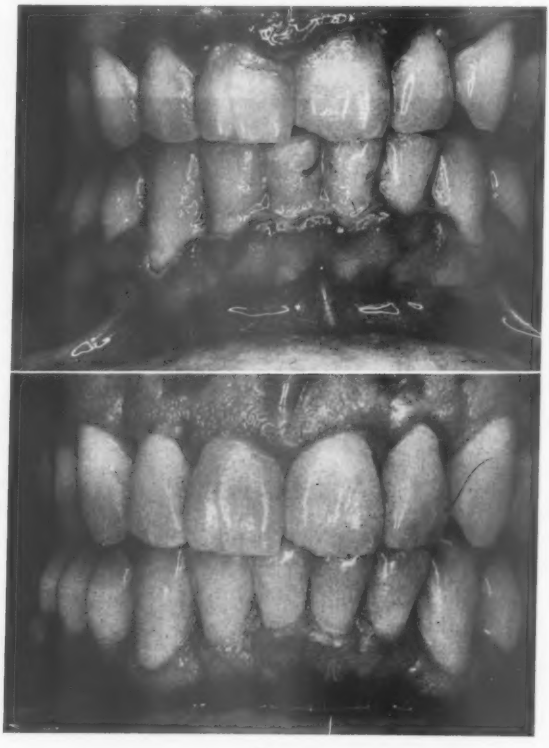
Fig. 15.—A Dilantin hyperplasia characteristically starting at the interproximal areas. Note the lack of overgrowth in the edentulous section.

Gingival Pathology.—The gingival margin may thicken and grow coronally as a result of either local inflammation or such systemic influences as Dilantin, idiopathic fibromatosis, or blood dyscrasias. Resultant dimensional changes are merely exaggerations of the basic gingival pattern with the most marked effects at the site of greatest normal growth, the papilla. It is here that inflammation occurs most frequently,[19] along with hypertrophies and hyperplasias.[2, 4, 24]

These hyperplasias disappear (Fig. 15), just as a normal papilla does when teeth are separated. The amount of separation, however, must be

Fig. 16.—Hormonal overgrowth on the labial surface rather than at the embrasure during pregnancy. This is an independent neoplasm of the gingival crevice rather than a simple swelling of the free gingiva.

A.



B.

Fig. 17.—*A*, Acute necrotizing ulcerative gingivitis with typical papillary destruction and formation of mesiodistal interdental clefts.

B, Postoperative recovery of papilla when a continuous surface of healing gingivae is achieved.

greater than in normal mouths in order to nullify the excessive gingival growth tendencies.

Nonspecific gingival inflammation during pregnancy shows the same growth behavior as any other inflammation. Pregnancy tumors do not follow this plan and may occur at any marginal location (Fig. 16), for they are independent pedunculated growths rather than simple swellings of the free gingivae.[25]

Necrotizing ulcerative gingivitis causes a destruction of the papilla with formation of a mesiodistal interproximal cleft. This produces independent buccal and lingual knobs of gingivae analogous to the situation of teeth in extremely close contact. When buccolingual continuity is restored, either by curettage alone or by curettage combined with surgery, the gingiva responds to the embrasure proportions. A papilla re-forms (Fig. 17, *A* and *B*) as long as there has been no great loss of epithelial attachment.

Epithelial Attachment Levels.—In the ideal situation the epithelial attachment pattern closely follows the physiologic gingival pattern, resulting in a shallow crevice. When attachment loss does not follow this pattern, periodontal pockets are formed. The greater the deviation between attachment pattern and gingival pattern, the deeper the pocket.

Fig. 18.—Steep vertical gingival gradients resulting from toothbrush abrasion. The more prominent tooth surfaces are the sites of greatest abrasion and recession.

Gottlieb[8] wrote that a flat pocket bottom would more likely foster gingival recession than would a deep precipitous one where the high points of attachment on either side would presumably prevent shrinkage. While this seems logical, it does not account for the countereffects of gingival growth tendencies.

It is true that a flat attachment pattern would be likely to produce a shallow crevice where the natural gingival contour is flat, as on the buccal surface of a molar, but the same pattern on a proximal surface would establish a deep pocket under the crest of the papilla. This was borne out in a previous study[13] in which flat or slightly concave interproximal pocket bottoms

produced no shrinkage after resolution of inflammation. Furthermore, a precipitous epithelial attachment pattern on the labial surface of a prominent cuspid would probably create a recession rather than a pocket.

Gottlieb's concept could be restated as follows: The attachment level pattern least likely to produce a pocket is the one that is parallel to the *physiologic gingival pattern*. When these two patterns diverge, the most coronal point of attachment establishes the level at which a *physiologic gingival pattern* is formed (Fig. 14, *HEA*).

Artificial Abrasions.—Recessions caused by the abrasion of toothbrushes or other foreign objects are well known. Since the most prominent tooth surfaces quite naturally receive the most abrasion, the most apical sections of the gingival margin are shifted further apically. The result is a more extreme gradient as the gingiva climbs toward the papilla (Fig. 18).

CHANGING THE FOSITION OF THE MARGIN OF THE GINGIVA

Pocket elimination, except in the infrequent instances of reattachment, is an intentionally created permanent recession where epithelial attachment is close to and parallel to the gingival margin. A knowledge of the basic and modifying factors affecting gingival position acts as a guide in reaching this goal.

Gingival Pathology.—Since one cannot predict the exact degree to which an individual mouth conforms to these principles, it is sound practice to start with treatment of the basic gingival pathology before more involved, traumatic procedures are deemed necessary. In the great majority of cases this means meticulous root surface curettage and education of the patient in oral hygiene. Therapy stops at this point if shrinkage creates the desired effect.

Tooth Surface Accessibility.—Crown positions may be altered by many techniques, although there are simple methods[1, 11, 20-22] especially adapted to the problems of periodontal orthodontics.

Crown forms are modified by reducing exaggerated buccal, lingual, and proximal contours. This usually has much less effect on gingival topography than do orthodontic methods. The most important site for coronal reshaping is the gingival embrasure. However, it is often technically impossible to modify adjoining metal surfaces or solder joints appreciably. The choice is either replacement of the filling or bridge or surgical modification of the embrasure.

Where thick bone is considered responsible for gingival growth (Fig. 7, *A* and *B*), it is thinned and tapered, so that it ends as a knife edge at the crest. This is also done whenever the vertical height of bone is surgically reduced.

Epithelial Attachment Level Patterns.—Well-conceived surgical procedures use the greatest pocket depths (Fig. 14, *LEA*) as the level at which to establish a new *physiologic gingival pattern* (*PGP'* on molar). The removal of all attached and unattached periodontal structures (including bone, when present) coronal to this preplanned outline at once re-forms both gingival and epithelial attachment patterns to conform to accessibility gradients.

Compromise surgery is frequently indicated when the amount of support to be excised would endanger tooth stability. The degree of necessary compromise is also a measure of the residual penchant for gingival regrowth.

These surgical principles were described in part by Neumann[14] in 1912 and were re-established, expanded, and clarified in more recent times by Schluger,[18] Friedman,[3] and Ochsenbein.[16]

Simple removal of unattached gingivae is effective only when pockets are caused by gingival overgrowth superimposed on an intact ideal attachment pattern. Prevention of regrowth may involve treatment of metabolic disorders inciting this phenomenon.

The interproximal space has warranted repeated discussions above, and once again it deserves special mention. If the surgery on the molar in Fig. 14 were performed only on the buccal surface, the undisturbed height of the papilla would be the guide for re-establishment of the original gingival pattern. The height of the papilla must be reduced to form a harmonious stable relationship with the desired buccal recession.

Papillary height is reduced by evenly lowering proximal attachment levels and, if necessary, interseptal bone. This widens as well as lengthens the embrasure,[7] since roots are generally further apart at the more apical levels. With these new dimensions, the bordering tooth surfaces are more accessible and free gingival growth is less marked. This is the method used when proximal tooth reshaping is ineffective (Fig. 11, *A*, *B*, and *C*).

The bony floor of the embrasure may be lowered either for the foregoing reasons or for elimination of interproximal concave pockets by diminishing the buccal and lingual plates. In any case, when the floor is reduced sufficiently, it may be apical to the level of the adjacent buccal or lingual gingivae. To prevent undesirable interproximal gingival regrowth, the new *physiologic gingival pattern* must be extended by reducing the buccal or lingual gingivae.

Thus, to eradicate a deep buccal pocket effectively it may be necessary, in turn, to remove buccal, interproximal, and lingual tissue.[15]

Artificial Abrasions.—Recessions caused by foreign object trauma are stopped by education of the patient. This same abrasive action is used therapeutically with rubber tip or other devices to inhibit gingival growth where ideal surgery is not possible or where the growth is excited systemically, as in the case of Dilantin.

SUMMARY

Although the exact mechanism is unknown, certain empirical correlations make it possible to predict changes in the position of the free gingival margin. This information is useful in attaining pocket elimination and preserving periodontal health.

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ORAL MEDICINE

PROGERIA

Report of a Case

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INTRODUCTION

Progeria is a rare disease of childhood first described by Hutchinson[1] and Gilford.[2] Children with this disease basically exhibit (1) a marked reduction of growth in size, without any significant arrest of bone maturation and with retained infantile characteristics, and (2) senile changes affecting the cardiovascular system and the distribution of subcutaneous fat. Elaboration of these symptoms includes dwarfism, almost complete baldness, beaked nose, receded chin, narrow chest, prominent veins, arthritic changes, and atrophy of skin and subcutaneous tissues with the skin showing brownish pigmented areas.[3]

In the literature on progeria,[4-8] little has been discussed concerning the dental condition found in these patients or the radiologic interpretation of the individual bones of the body. It is hoped that this case report will lead to a better understanding of the over-all curiosities associated with progeria.

CASE REPORT

Chief Complaint.—A 10½-year-old white boy with a previous diagnosis of progeria was readmitted to the Children's Hospital of Philadelphia on March 11, 1957, because of discomfort in the mouth secondary to dental caries.

Past Medical History.—At birth, the patient was a normal 6½ pound male infant, delivered without instrumentation on Sept. 19, 1946. His birth height was normal, and at 7 weeks he weighed 10½ pounds. At this age it became apparent that the child would not move his left leg, which he kept externally rotated and flexed. He screamed in pain when handled. He was hospitalized for more than one month and was treated with penicillin following a diagnosis of acute arthritis of the left hip. Penicillin therapy was not effective, and the joint was aspirated. The condition gradually improved. Upon discharge from the hospital, the patient appeared weak and weighed only 7 pounds.

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He sat up at 7 months. At 9 months he lost what had appeared to be a healthy crop of hair, a common finding in this condition. He began to talk at 13 months. When the child first walked at 14 months, his joints seemed "stiff." At 15 months the fingernails and toenails were hard, yellow, and brittle. The skin seemed to be thin and taut. When the patient was 2 years of age, a diagnosis of progeria was established at the hospital of the

Fig. 1.—Photograph of the patient at 10½ years of age showing typical features of progeria, with a height of 40 inches and weight of 31½ pounds.

University of Pennsylvania. At 4 years his blood pressure was 95/50, his height was 37 inches, and he weighed 25 pounds (the normal mean height and weight being 40.9 inches and 38.1 pounds).[10] He tired easily, but he was mentally alert. He played with other children but was inclined to be timid.

Family History.—The patient's mother and father were both living and normal. There was no history of any similar condition in either family's background. Two younger siblings were perfectly normal.

Present Illness.—Because of a severe dental condition and the inability to masticate adequately, the patient was referred to one of us for treatment. The child actually had no complaints other than his dental problems and the knowledge that he was very different from other children.

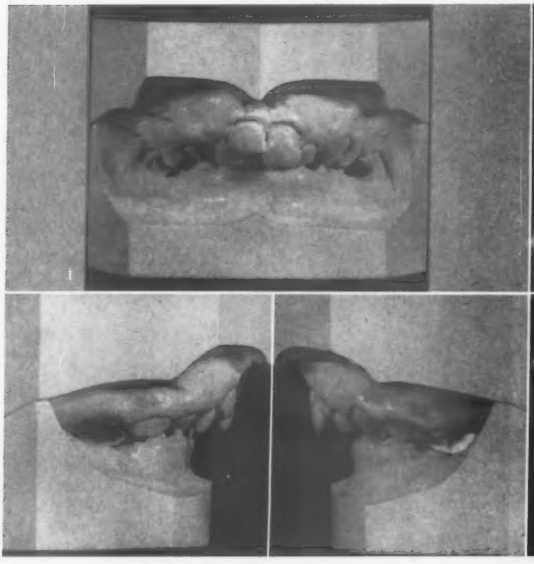
Fig. 2.—Photograph of head showing prominence of superficial veins, sparseness of hair, closely spaced eyes, slender nose with a birdlike appearance of maxilla and absence of eyebrows.

Fig. 3.—Photograph of hands showing the skin to be dry, thin, taut, and mottled. The fingers are small with short, brittle, dry, and broken nails. All the joints appear unusually prominent.

Physical Examination.—Physical examination revealed a dwarfed 10½-year-old white boy with the physical features of an old man (Fig. 1). His height was 44 inches and his weight was 31½ pounds (as compared to the normal means of 55.5 inches and 76.8 pounds).[10] The hair of the scalp and body was absent except for fuzz in the suboccipital region. There were no eyebrows (Fig. 2). The skin was dry, thin, and mottled

with brown pigmented areas. The superficial veins of the scalp were prominent. The eyes were closely spaced, and the patient wore glasses for hyperopia. The nose was slender and "birdlike." The mandible was underdeveloped. The head appeared large, but this was only because of the appearance of a normal head on a small body. The heart and lungs were normal, with a blood pressure reading of 100/80. The abdomen was negative. The rectum and genitals were normal, with testes descended. All of the extremities were small in proportion to the trunk. The fingers and hands were small with very short, brittle, dry, and broken fingernails (Fig. 3). The knees could not be fully extended, and the ankles showed limitation of motion. He walked with a stiff-legged gait. Normal foot pulses were palpable on each side. There was surprisingly good muscle power in all extremities, with no tremors. All deep and superficial reflexes were normal. The cranial nerves were all intact. The voice was high pitched and weak.

Laboratory Findings.—Laboratory studies revealed the following: serum calcium, 10.3 mg. per cent; serum phosphorus, 4.7 mg. per cent; phosphatase, 3.7 Bodansky units; and nonprotein nitrogen 14.0 mg. per cent.



A. B. C.

Fig. 4.

A. B.

Fig. 5.

Fig. 4.—Marked retrusion of the mandible producing limited oral space when maxilla and mandible are opened.

Fig. 5.—Models of maxilla and mandible showing arrangement of primary teeth as well as broken-down condition of mouth. The mandibular permanent central incisors have erupted lingual to firmly attached primary incisors. These are the only teeth which have erupted in this 10-year-old boy.

Dental Examination.—The patient presented a badly broken-down mouth. The mandible was receded symmetrically, producing a small area or space between the maxilla and the mandible (Fig. 4). This made it extremely difficult for the patient to open his mouth to allow a dentist to perform adequate dentistry. The mandible showed a short rámus, and the angle of the ramus was obtuse. The tongue was small but otherwise normal. The maxillary arch was narrow with a high vault. The deciduous teeth were yellowish brown and deeply stained; they had the parallel appearance of teeth seen in

old persons. This youngster, at 10½ years, showed almost a full complement of deciduous teeth. The permanent mandibular central incisors had erupted lingual to their deciduous predecessors. No other permanent teeth had erupted (Fig. 5). The deciduous

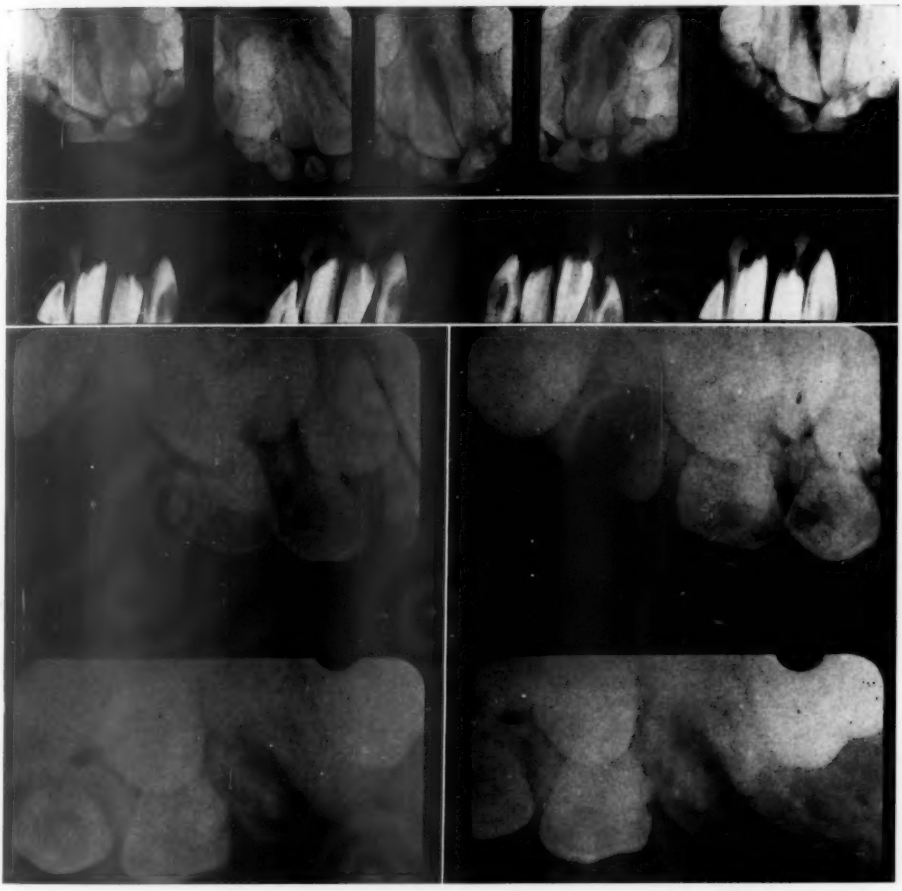
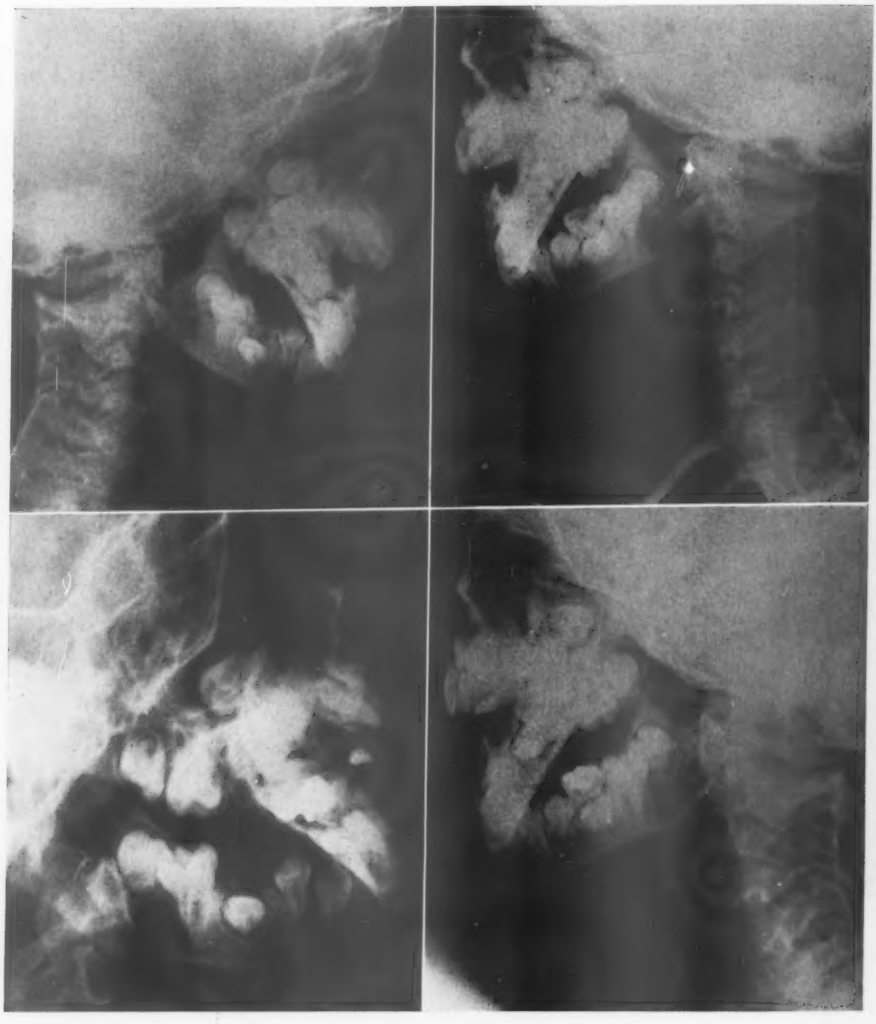


Fig. 6.—*A*, Roentgenograms of maxillary and mandibular incisor area. Maxillary central incisor roots are almost fully formed. Note retention of permanent teeth, which is associated with condition of cleidocranial dysostosis. *B*, Retained roots and carious maxillary posterior teeth.

teeth, especially the anterior ones, were firmly attached to the tissues. At this age, the first permanent molars, the upper and lower incisors, and the first premolars should have erupted.

Dental Roentgenographic Examination.—The maxilla and the mandible showed the permanent teeth present in their respective regions (Fig. 6). The lower first permament molar roots were almost fully formed, the normal completion time being 9 years of age. The upper incisors were approaching completion seen at the age of 10 years. The upper lateral incisors were following their dental-root timetable. The lower second permanent molars appeared radiographically to indicate the age of 9 years and were located well

A. B.



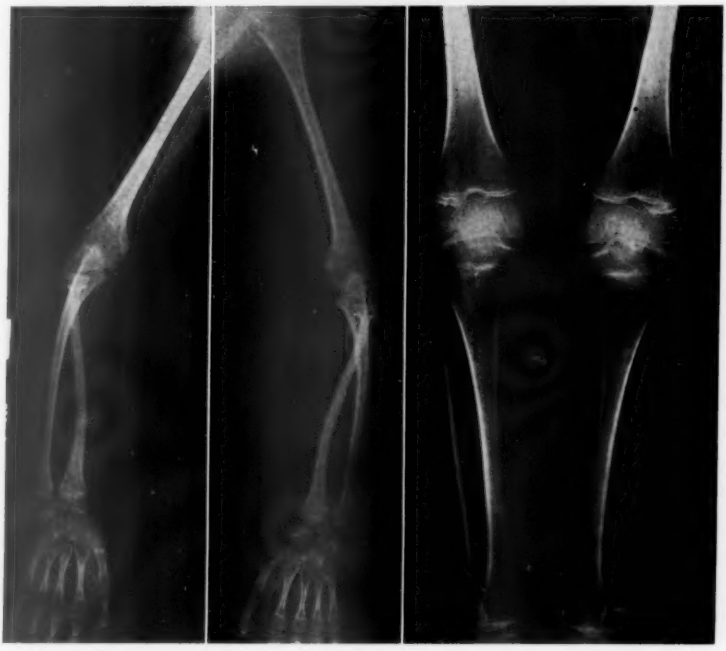
C. D.

Fig. 7.—Oblique roentgenograms of right and left sides of mandible showing mouth before and after dental work. Observe unerupted permanent molars and premolars as well as their crown and root development.

up in the ramus of the mandible. The lower canine root gave the chronological picture of 10 years. This root was close to the lower border of the mandible and presented the risk of a fracture of the mandible in the event any extraction should be indicated. The other teeth were slightly behind their chronological age. No evidence of third molars was present. The pulp chambers and root canals of the deciduous teeth appeared to be narrower as a result of secondary dentine formation.

Skeletal Roentgenographic Examination.—A comprehensive radiologic examination of the skeleton was performed. In general, no gross disturbances of development were noted, but there were a number of small findings consistent with other reported cases of progeria.

The long bones (Fig. 8) were slender and delicate, but there were no gross structural abnormalities. (Note the extreme thinness of each fibula in Fig. 8.) Because of the slender, delicate long bones, the knee and elbow joints appeared unusually prominent (Fig. 9). The capitellum of each elbow was unusually large with a widened epiphysis of the head of each radius. An extreme degree of coxa valga was present, as shown in the roentgenogram of the pelvis (Fig. 10).



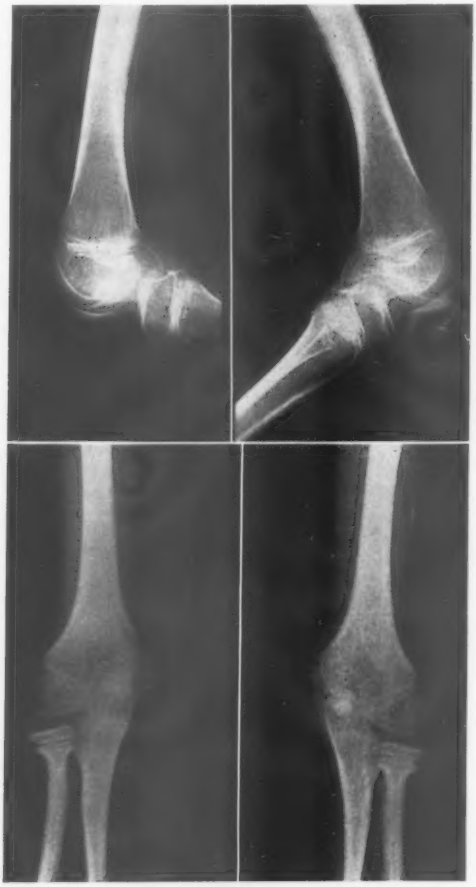
A. *B.*

Fig. 8.—The long bones are all slender and delicate. This is most pronounced in each fibula (*B*). The knee and elbow joints appear unusually bulky, but this is in contrast to the slender shafts.

The most abnormal finding of the skeleton was seen in the terminal phalanges of the hands and feet. The photograph of the hands shows the ends of the fingers to be unusually short with very small fingernails (Fig. 3). The roentgenograms of the hands and feet (Fig. 11) show the reason for this: Although the metacarpals and metatarsals and the first and second phalanges of both feet and hands were normal, all the terminal phalanges were abnormally short and tapered abruptly to a pointed end.

The thoracic cage (Fig. 12) revealed slender, delicate ribs. There were partially healed fractures of the posterior portion of the right third and fourth ribs where the patient had recently sustained a blow. The clavicles were extremely slender and shortened at their distal ends. The cardiac silhouette was within the normal limits of size.

A.



B.

Fig. 9.—These views of the knees and elbows give a better detail of their size relative to the slender shafts. Note the large capitellum in each elbow. Note also the way in which the epiphysis of the radial head has grown to accomodate the larger capitellum (*B*).

The spine (Fig. 13) showed nothing abnormal.

The roentgenograms of the skull (Fig. 14) showed the bones of the cranial vault to be thin with a very narrow diploic space. All the sutures were still open and showed the normal degree of closure that one would expect at this age. There was still an opening in the region of the anterior and posterior fontanels. The hypophyseal fossa appeared normal. The posteroanterior view of the skull revealed the reason that the

face appeared "birdlike." The maxilla was markedly narrowed in relation to both the face and the mandible. The lateral film of the skull shows the short mandible, and in this view the maxilla overhangs the mandible. The angle made by the vertical and horizontal rami of the mandible measured 155 degrees. This angle was very obtuse when compared to the normal angle of about 120 degrees. From the lateral view, one could see the marked craniofacial disproportion with the small facial bones attached to a normal-sized vault. The mastoids were poorly developed for a 10½-year-old child, as seen in the occipital view. The mastoid films (Fig. 15) were taken when the boy was 6 years of age. These were infantile with no cellular development.

Fig. 10.—The pelvis shows a severe degree of coxa valga, bilateral. The apparent calcifications over the right ilium are retained opaque in the bowel. The epiphysis of each lesser trochanter has appeared.

There was no advancement of bone maturation as shown by the epiphyseal development of the hands and wrists, elbows, knees, and pelvis. The bone maturation was that of a 10½-year-old boy. There was fusion of the epiphyses of the terminal and middle phalanges.

Dental Procedure.—Operative dentistry was performed under general anesthesia in the hospital operating room. When the primary molar teeth were prepared for filling, it was found that the pulp had receded considerably, as in old age, and the dentin did not appear to have the vitality seen in youthful teeth. The cavity floor was of more than normal depth before firm dentin was reached. Occlusal fillings were placed in the upper right and left first primary molars and the lower right second primary molar. Extractions were done where indicated.

When the patient was seen four, six, and eight months postoperatively, the areas where extractions had been performed still had not healed completely.

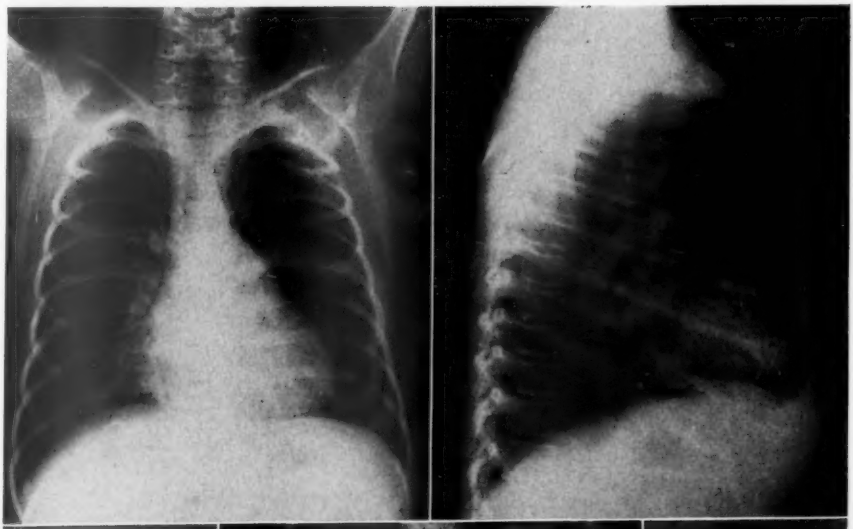
Histologic Examination.—The over-all histologic sections showed great irregularity in size and shape of the odontoblasts, with an increasing reduction in their number toward the apical end, where reticular atrophy was most prominent. While vascularization and the degree of hyperemia did not deviate from what is considered normal, there

A.

B.

Fig. 11.—The terminal phalanges of both the hands and feet are short and taper abruptly to a pointed end. This is best shown in each great toe (*B*). There is fusion of the epiphyses of the terminal and middle phalanges.

A. Fig. 12. B.



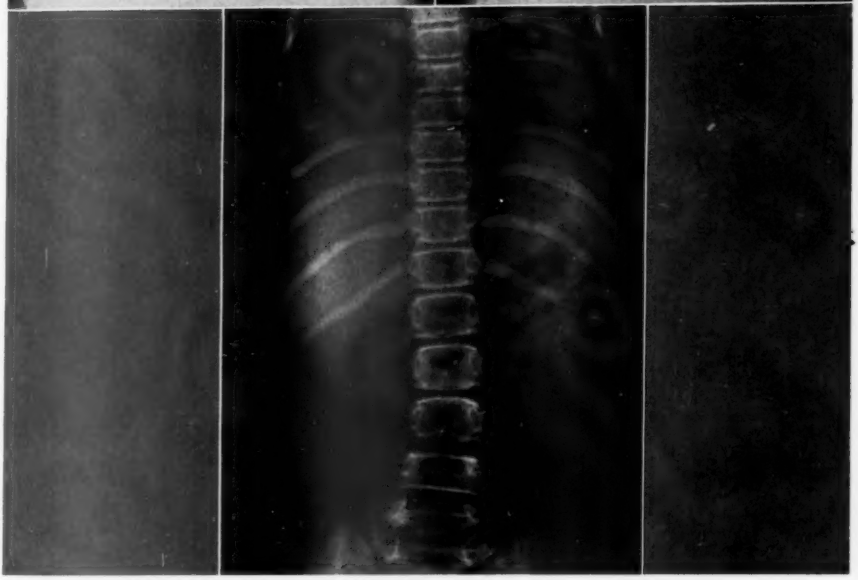


Fig. 13.

Fig. 12.—The thoracic cage reveals slender, delicate ribs with partially healed fractures of the posterior portion of the right third and fourth ribs. The clavicles are very slender and shortened at their distal ends.

Fig. 13.—The spine shows nothing abnormal. There is a minimal scoliosis, but this is due to position.

A. B.

C.

Fig. 14.—All the views of the skull show relatively thin bones of the vault with a very narrow diploic space. The occipital view (*B*) shows that a definite opening still persists in the region of the posterior fontanel. The posteroanterior view (*A*) shows a small, narrow maxilla. The lateral view (*C*) shows a short mandible and the pronounced overhang of the maxilla. The rami of the mandible make an obtuse angle of 155 degrees. The entire face appears small in relation to the normal-sized cranial vault.

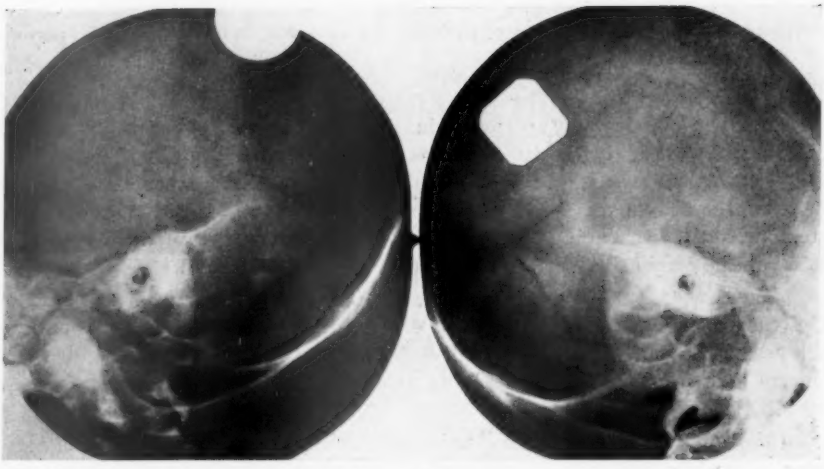


Fig. 15.—Oblique views of the mastoids taken in 1953, when the patient was 6 years old. Each mastoid is infantile with no cellular development.

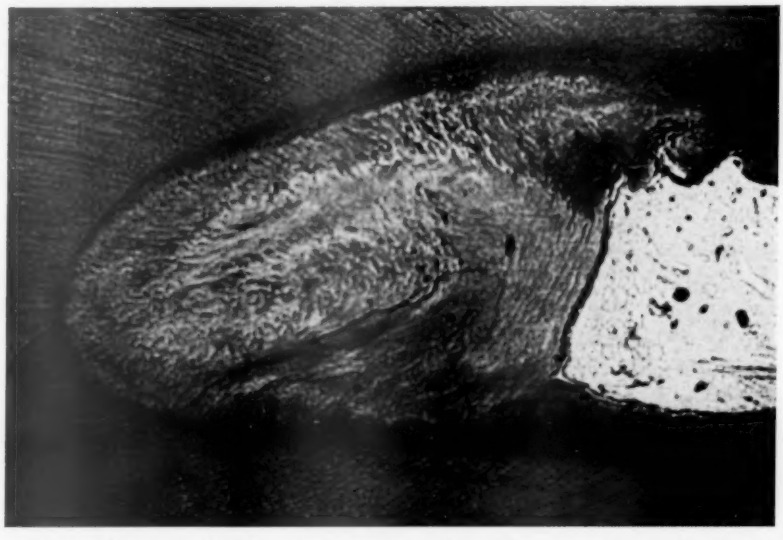


Fig. 16.—The coronal portion of the central incisor shows unusual amounts of irregular secondary dentine beneath which pulp cells appear atrophic. In the central area of the section, pyknotic odontoblasts beneath the secondary predentine have assumed a squamous shape.

was a diffuse calcification of varying degrees, from isolated minute calcospherites to larger conglomerations of calcium salts along the nerve fibers and the vascular walls. The irregular width of predentin was equalled by the variation of cementum at the periphery. Osteoclastic resorption of moderate degree was observed at the apical portion.

DISCUSSION

This is a classical case of progeria. The patient was apparently a normal child at birth, but by the time he was 4 months old it began to be apparent that he was underweight for some unknown reason. By the age of 1 year most of the diagnostic signs of progeria were present. In order to differentiate progeria from aging associated with certain types of mental deficiency, it should be emphasized that his mental state has been normal.

We have no explanation for this disease, and we are simply presenting the case as a comprehensive study of an interesting and little understood disease. Talbot and his associates[11] believe that these children suffer from an inborn error of metabolism involving an enzyme of importance in the energy-metabolism cycle.

Most of these children die prior to adolescence of coronary artery disease. To date our patient has had no symptom referable to his cardiovascular system. More of these cases must be reported before we can better understand the true etiology of progeria.

Acknowledgment and appreciation are given to Dr. M. Michael Cohen, Boston, Massachusetts, for the preparation of the histologic sections, and to Mrs. Lisabeth Baumann, Assistant Professor of Histology and Oral Pathology, School of Dentistry, University of Pennsylvania, for her kindness in reading and evaluating the histologic sections.

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PHARMACOLOGY AND THERAPEUTICS

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A NEW AGENT FOR SHORTENING RECOVERY TIME IN ORAL SURGERY

Ralph O. Christensen, D.D.S., Minneapolis, Minn.

HASTENING the recovery of patients from anesthesia is extremely important to the oral surgeon and his staff from the standpoints of economy and of easing or reducing their work load. Any preparation which could perform this function would be an extremely worth-while addition to the oral surgeon's armamentarium.

Recently we were introduced to parenteral Ritalin,* a central nervous system stimulant which rapidly and safely overcomes the effects of Pentothal, Surital, Neraval, and similar anesthetics. Parenteral Ritalin produces improvement of vital functions with lessened depth of coma and prompt return to responsiveness with little or no after effects.

Meier and associates,[1] as well as other clinicians,[2-4] have demonstrated that Ritalin has specific central stimulating properties with minimal effects on heart rate. In their work, Meier and his co-workers[1] also noted that parenteral doses of Ritalin distinctly lessened the anesthetic and hypnotic effects of Pentothal sodium. In another study, Plummer[5] found that Ritalin had an antagonistic effect on reserpine in dogs, rabbits, and monkeys, which was later confirmed in human beings. Other studies have shown that the drug has a wide margin of safety with little or no effect on pulse, blood pressure, urine, or liver and kidney function.

Moreover, in extreme cases, where full consciousness is not immediately restored, marked improvement in respiration and cardiovascular status has been noted.

CLINICAL STUDY OF 560 PATIENTS

We conducted a clinical trial with this agent in over 560 patients. They ranged in age from 7½ to 75 years. No attempt was made to select patients according to sex or type of operation performed. Ritalin was used as an

*Ritalin (methylphenidate hydrochloride CIBA) was supplied by CIBA Pharmaceutical Products, Inc., Summit, New Jersey.

"awakening" or recovery agent in surgical procedures of every variety encountered in a dental surgical office (removal of impactions, osteomas, and cysts, extractions, multiple extractions, alveolectomies, etc.).

PROCEDURE

After blood pressure, pulse, etc., were taken, we gave 1/150 gr. atropine as a preanesthetic agent prior to inducing anesthesia (Neraval, Surital, Pentothal, etc.). Ritalin was injected immediately postoperatively. In this study, dosage of Ritalin ranged from 20 mg. to 100 mg. Our average dose was 20 mg.

With Ritalin, the majority of our patients were out of the recovery room and office within thirty minutes, although recovery times ran from one minute to two hours (in one case). In our opinion, Ritalin reduced the usual recovery times by about one-half.

We encountered no case of nausea in our series of 560 patients, although the possibility of nausea should always be considered in patients who are hyperactive, no matter what anesthetic or drug is used.

The following two cases are illustrative of our experience with Ritalin:

CASE 1

Patient S. N., an 18-year-old boy in good physical condition, presented on Dec. 21, 1956, for removal of upper and lower impacted right third molars. The impacted teeth were removed under thiopental sodium anesthesia, with 700 mg. of 2½ per cent solution being used. The time of operation was approximately fifteen minutes. Recovery time was approximately one hour.

The same patient presented again on Sept. 27, 1957, for removal of impacted upper and lower left third molars. At this operation, 750 mg. of 2½ per cent thiopental sodium was used for anesthesia. Time of operation was approximately thirty minutes. At the conclusion of surgery, 20 mg. of Ritalin in 2 c.c. of distilled water was given intravenously.

The recovery time was noticeably shortened, being approximately one-half hour. It will be noted that 50 mg. more of anesthetic solution was used at the second operation and the length of operating time was almost doubled, but with the administration of 20 mg. of Ritalin the recovery time was reduced by one-half.

CASE 2

Patient L. S., a 65-year-old woman with a condition of lymphatic leukemia presented on July 10, 1956, for the removal of a lower right abscessed second molar tooth. The anesthesia used was 500 mg. of 2½ per cent thiopental sodium. The recovery time was approximately one hour.

On Nov. 18, 1957, the patient returned for the removal of an abscessed upper right first molar. At this operation, 500 mg. of 2½ per cent thiopental sodium solution was used. The time of operation was identical for both procedures, and the amount of anesthetic solution being used was identical. However, at the conclusion of the second operation, 30 mg. of Ritalin was given intravenously in 3 c.c. of distilled water and the recovery time was reduced to approximately one-half hour.

DISCUSSION

I have been practicing oral surgery for over seventeen years and have used such anesthetics as Pentothal, Surital, Neraval, etc. in more than 10,000

cases. Bringing patients rapidly and safely back to consciousness after anesthesia has long occupied my thinking, since more often than not recovery delays run to an hour or more with their attendant drain on office time and space and with discomfort to the patient. The introduction of Ritalin, a central nervous system stimulant which rapidly overcomes the effects of anesthesia, seems to be the answer to this problem.

Although I have an excellent anesthetic team in my office, Ritalin has proved extremely valuable to us, both economically and practically, and we now use it routinely. It has reduced our recovery time by one-half and has eased our work load tremendously, since patients move out of the office rapidly and under their own power. Moreover, because of Ritalin, they are more alert on leaving the office, and we have little fear of their not getting home safely.

We have many patients returning from year to year, and in almost every instance since we have been using Ritalin we have noted not only a shortening of the recovery time but also a much more rapid return of muscular coordination and a lessening of the euphoria so common following the use of intravenous barbiturate anesthesia.

We have also found that Ritalin has actually increased the length of our operative day. Before its use, we never took a patient after 4 P.M., since a recovery delay would mean a one- to two-hour wait. With Ritalin we are now fairly certain that a patient operated on at 4 P.M. will be ready to leave the office under his own power by 4:30 or 5 P.M.

There were complaints of sleeplessness in a few patients receiving higher doses of Ritalin (40 to 50 mg.) late in the day. It is suggested, therefore, that minor operations, where lesser amounts of anesthesia are used, should be scheduled for the later hours of the surgeon's day.

SUMMARY AND CONCLUSIONS

We used parenteral Ritalin, a safe new central nervous system stimulant, to hasten postanesthetic recovery in over 560 patients and had extremely good results. With Ritalin, recovery was earlier and better, and there was no relapse of drowsiness. There was also a much more rapid return of muscular coordination and a lessening of the euphoria so common following the use of intravenous barbiturate anesthesia. In general, patients were up and out of our office within twenty-five to thirty minutes. Except for insomnia in a few cases where higher doses (40 to 50 mg.) were given late in the day, we encountered no side effects.

We found Ritalin very effective as a postanesthetic "awakening" agent, although we did note one or two patients who did not seem to respond to the drug. We recommend further study of this phenomenon, since immediately repeating the dose did not change this lack of response.

It is our personal opinion that Ritalin is an excellent agent for rapid, safe postanesthetic recovery. We highly recommend its routine office use to every oral surgeon. It is an excellent means for reducing staff work load,

minimizing the possible hazards of postanesthetic side effects (drowsiness, headache, depression, etc.), and clearing the office rapidly. The use of Ritalin should prove time saving and economical.

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Comment by H. W. Krogh, Washington, D. C.

There is as yet no direct drug antagonist to the barbiturates, although diligent search is being made to find one. Those substances which are said to counteract barbiturate overdosage or speed up barbiturate degradation produce their effects by stimulating the central nervous system and perhaps indirectly increasing metabolic rate and *not* by direct action against the barbiturate. Some of these drugs are shorter acting than the barbiturates, so that the awakening may be of short duration, with the patient relapsing into a deep sleep. Others are longer acting than the barbiturates and, if used to awaken quickly patients who have had a small total dose of barbiturate, produce prolonged mental stimulation which interferes with normal sleep. Finally, it is well to remember that all analeptics are central nervous system stimulants and thus increase oxygen demand in the brain. Accordingly, they should not be used in the presence of an oxygen deficit which cannot readily be corrected.

It is to be hoped that the history of the analeptics will not repeat that of the antibiotics and muscle relaxants. Specifically, it is hoped that analeptics will not be used to cover up careless technique or as substitutes for sound anesthesia practices.

Comment by Floyd L. Paynter, D.D.S., Omaha, Neb.

I have reviewed the clinical reports on parenteral Ritalin, the article written by Dr. Christensen, and the comments of Dr. Krogh.

As Chief of the dental section of the staff at our new Clarkson Memorial Hospital here in Omaha, I have had the opportunity to work with general anesthesia, and we have used this material somewhat. Naturally, I feel that any comment I could make should be backed up by something out of my own experience rather than from what I have read in the literature.

We have used Ritalin on ten patients. The dose in two cases was 10 mg. Ritalin intravenously. The last case, on March 5, received 350 mg. of Pentothal sodium for a D. & E. The patient was given 10 mg. Ritalin intravenously. Her blood pressure was 110/70, and after Ritalin it went up to 170/110. The patient aroused quickly and was rational within fifteen minutes.

Two cases showed a rise of 30 to 40 mm. Hg in blood pressure following intravenous Ritalin. I think that the drug has excellent merit, but I feel that it should be given slowly or in small doses and repeated. One feature not noted with Ritalin is that disadvantage seen with picrotoxin, Metrozol, and others of the analeptic group, namely, a relapse into the sedated state after initial stimulation and responsiveness.

Dr. John Adriani, one of our most eminent anesthesiologists from Charity Hospital in New Orleans, has recently published a paper in which he states that the drug has good possibilities.

ORAL ROENTGENOLOGY

A SIMPLE METHOD FOR THE X-RAY LOCALIZATION OF THE UPPER IMPACTED CANINE

Hermine Nitsche, M.D., and Edith Vályi, M.D., Budapest, Hungary

THE roentgenographic localization of the upper impacted canine is desired when orthodontic treatment or surgical removal of the tooth becomes necessary.

There are many ways in which this can be accomplished. Ivy,[4] Kaufman,[5] and others have published articles dealing with this problem, but their

Fig. 1.

Fig. 2.

Figs. 1 and 2.—The central ray enters from the opposite side, at the root of the nose.

From the Stomatological Clinic of the University of Budapest (director, Prof. Charles Balogh).

methods involve taking several radiographs and making a diagnosis by comparison of the pictures. Taking the series of radiographs, as well as comparing and evaluating them, takes a lot of time. We endeavored to find a quick, simple, yet reliable method. A description of our technique follows.

SINGLE X-RAY TECHNIQUE

A 9 by 12 cm. occlusal film is placed between the upper and lower teeth, not in the middle but nearer to the examined side. The patient bites on it gently in order to fix it. The patient's head is upright, with the nose and mouth in the median plane, so that the film's position is horizontal. The tube is directed toward the supraorbital foramen on the opposite side, namely, to the root of the nose, at an angle of 55 degrees to the horizontal plane and at 10 degrees to the frontal plane. Exposure time is three seconds for 45 kv. (Figs. 1 and 2).

Fig. 3.—Impacted upper left buccal canine. The projection gives the impression of a palatal impaction.

CONCLUSION AND SUMMARY

With this contralateral alignment, we get a distorted but separated picture of the superimposed teeth. The chief advantage of our method is that the buccally impacted canine never appears on the palatal side of the teeth, as it may with half-axial projections (Fig. 3). One single picture gives accurate localization. All data concerning the exposure are simple. No meditation or comparison of several pictures is needed; a simple inspection of our films is sufficient (Figs. 4 and 5).

Fig. 4.—The distorted picture reveals the real situation: a buccal location of the canine *(C)*.

Fig. 5.—Left upper canine appears on the palatal side. The right upper canine is palatinally impacted too.

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ORAL PATHOLOGY

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ASPIRATION OF CELLS INTO DENTINAL TUBULES?

Harold R. Stanley, Jr., D.D.S., M.S., B.S., and Herbert Swerdlow, B.A., D.D.S., Bethesda, Md.

OPINIONS differ regarding the cause of odontoblasts and blood cells in dentinal tubules. When we observed odontoblasts mixed with inflammatory cells and erythrocytes in dentinal tubules beneath a cavity preparation, we felt that the phenomenon probably was the result of crowding of the pulpal tissues, a build-up of intrapulpal pressure in a confined area by edema, hyperemia, and exudation. Since the odontoblasts were apparently pushed outward into the tubules by an internal force, we suggested that the word "displaced" was more appropriate than "aspirated."[1]

Although Orban[2] did not use the term "aspirated" in his original paper in which he demonstrated that compression of the root surface by the beaks of forceps was associated with the presence of odontoblasts in the dentinal tubules beneath the crushed cementum, the use of the term came into vogue at a later date, possibly to supplement Orban's implication that some pumping or sucking action might be responsible for the phenomenon. Apparently the term appeared in the English literature for the first time in a paper by Kramer and McLean,[3] who used this term to describe odontoblasts associated with forceps trauma as well as with cut tubules beneath experimental cavity preparations filled with self-curing acrylic resins. Consequently, in recent years the word has been applied rather loosely to describe any cell that might be found in tubules, irrespective of the actual cause of the phenomenon.

In this article additional evidence will be presented to strengthen the concept that the phenomenon, unless induced by forceps, is the result of increased intrapulpal pressure due to an inflammatory response.

Although leukocytes in dentinal tubules were previously described by Römer[4] in 1909 and Fisher[5] in 1939, Orban,[6] in 1940, was the first to provide an elaborate account of their presence. He stressed the point that an acute inflammatory reaction was always present in the pulp adjacent to the tubules

From the National Institute of Dental Research, National Institutes of Health, Public Health Service, U. S. Department of Health, Education, and Welfare, Bethesda, Maryland.

containing leukocytes and suggested that the latter were attracted into the dentinal tubules by a chemotactic response resulting from a protoplasmic alteration of the odontablastic processes due to irritation.

In 1941, Orban[2] noted well-circumscribed areas of the pulp containing few cells where odontoblasts had formerly existed. He again observed within the calcified dentinal tubules elongated nuclei which he recognized as those of odontoblasts. He conjectured that the odontoblastic processes had shrunk as a result of the surface injury created by the forceps and had pulled their corresponding cell bodies and nuclei into the tubules, or that a slight compression of the dentine and pulp had produced some sucking or pumping action upon the odontoblasts. Fig. 1 shows a typical example of forceps-induced displacement as described by Orban.

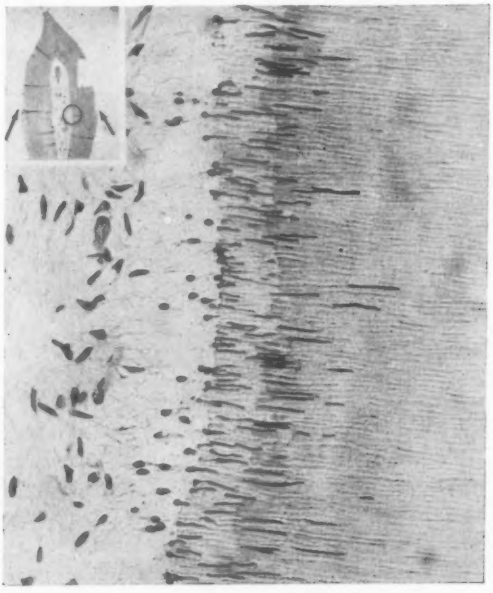

Fig. 1.—Photomicrograph of 107-day, 150,000 r.p.m. specimen. Insert shows imprints of forceps (arrows) below cavity preparation. Enlargement reveals denuded areas in the odontoblastic layer and a few remaining isolated odontoblasts. Forceps displacement remains relatively shallow. (Magnification: enlargement ×280, insert ×2.5; reduced ⅙.)

Müller,[7] in 1948, considered odontoblasts in tubules to represent a form of pulpal degeneration. Rushton,[8] in 1949, observed erythrocytes in dentinal tubules close to severe pulpal hemorrhages resulting from trauma. In referring to Orban's works, however, he states that one could infer that the presence of leukocytes in the tubules was caused in a manner similar to that of the odontoblasts, perhaps sucked into the tubules on release of forceps pressure.

In 1952, Kramer and McLean,[3] using a slow-running handpiece with a No. 5 rosehead burr and a water spray followed by insertion of self-polymerizing

acrylic materials, found partial or complete disappearance of the odontoblasts at the pulpal ends of those dentinal tubules opened during cavity preparation. The missing odontoblasts could readily be demonstrated at varying distances in the tubules. They labeled this phenomenon "aspiration of odontoblasts." At first they thought that the presence of the cavity might in some way predispose to the aspiration of odontoblasts during extraction, but later they found that the phenomenon did not occur beneath cavities cut under comparable conditions and subsequently filled with zinc oxide and eugenol or coated by certain liquid cavity liners. They acknowledged that occasionally similar changes were observed in teeth filled by other methods but stated that a definite association existed between the aspiration and self-polymerizing acrylic material. Consequently, they put forth the following hypothesis: "When the acrylic resin filling is inserted into the cavity and the matrix band applied, the matrix will cover and seal off the free resin surface. When the critical phase of spontaneous polymerization is reached, the resin is expanding violently, the exothemic reaction is at its maximum. Free monomer vapor is trapped at the base of the cavity and exerts pressure on the contents of the dentinal tubules, so that the fluid is squeezed back into the pulp. The exothermic reaction of the resin subsides and tissue fluid flows back up the tubules, carrying with it the odontoblasts in the affected zone. Heat shock treatment of the resin will accelerate this process."

In 1952, Maeglin,[9] also working with self-polymerizing filling materials, described a range of "aspiration" that varied from slight invasion of the odontoblastic nucleus into the dentinal tubules to complete loss of cells from the odontoblastic layer.

James and Schour,[10] in 1955, studied the effects of cavity preparation and zinc oxide and eugenol and showed that, within the first hour after insertion of zinc oxide and eugenol, edema was prominent and the odontoblasts appeared to be clumped, some of their nuclei being seen in the tubules of predentine. In studying the effects of cavity preparation alone,[11] they observed within three hours a considerable number of odontoblastic nuclei within the tubules of predentine and the remaining odontoblasts crowded toward the predentine. After two weeks, granular debris, which apparently represented the fragmented remains of odontoblastic nuclei, was seen in the more peripheral portion of the dentinal tubules. It appeared to them that the presence of the odontoblasts in the tubules and the relatively slow recovery of the pulp were not related to the cutting procedures per se but to the failure to fill the cavity so that the pressure was greater within the pulp than within the unfilled portion of the cavity.

In 1956 we[1] studied the effect of a water coolant on human teeth while using a No. 37 inverted cone diamond stone at 20,000 r.p.m. All preparations were filled with zinc oxide and eugenol, and the teeth were extracted at intervals of from one hour to thirty-six days. By the end of one hour shallow displacement of numerous odontoblasts into the predentine or slightly beyond was observed in the presence of edematous changes. At this stage, however, the

odontoblastic layer was still generally intact and recognizable. At later intervals, if the remaining thickness of dentine beneath the cavity was greater than 0.8 mm., the histologic appearance of the pulps was the same in both the "with-spray" and "nonspray" categories. If the remaining dentine was less than 0.8 mm., however, there were histologic differences between the two categories. By the end of twenty-four hours, the "with-spray" specimens (Fig. 2) revealed disorganization of the odontoblastic layer with displacement of most of the odontoblasts and many blood cells into the dentinal tubules beneath the cavity preparations. Those few odontoblasts remaining behind were surrounded by leukocytes. A periodic acid-Schiff stain readily distinguished odontoblasts from

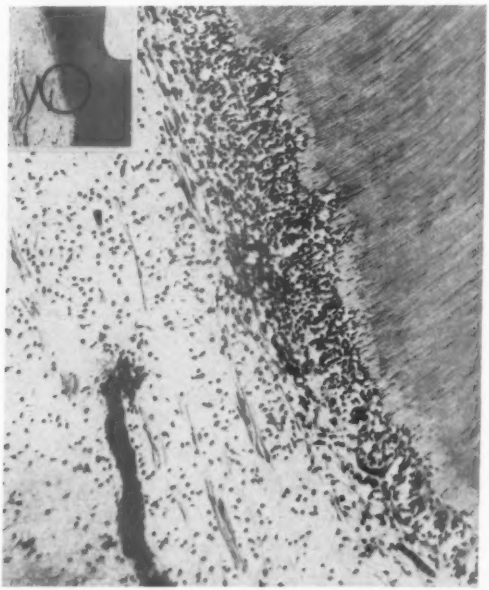

Fig. 2.—One-day "with-spray" specimen. A dense leukocytic infiltrate has for the most part replaced the odontoblastic layer. Note quantity of odontoblasts displaced into dentinal tubules. A large hyperemic capillary is seen in the deeper tissues. (Magnification: enlargement ×141, insert ×16; reduced ⅙.)

leukocytes in the dentinal tubules. The lesions were confined to the area directly beneath the cut dentinal tubules. In contrast, the "nonspray" specimens (Fig. 3) presented a burnlike lesion with prominent cellular degeneration, loss of cellular detail, and edema. Although many odontoblasts were missing from the odontoblastic layer, they could not all be accounted for on the basis of displacement. During the course of the experiment "with-spray" lesions remained confined to the region underlying the cut tubules and gradually underwent resolution, but in the "nonspray" specimens, in which burning apparently occurred, the lesions expanded beyond the cut tubules to involve the greater portion of the pulp. Whereas displaced odontoblasts were confined to the cut tubules in the "with-spray" specimens, they could be found beyond the cut

tubules and even on the opposite surface of the pulp (Fig. 4). The fact that expanding inflammatory lesions with displacement occurred in intact dentinal tubules of the pulpal wall opposite the cavity preparation led us to minimize the relationship of displacement to the type of filling material or grinding pro-

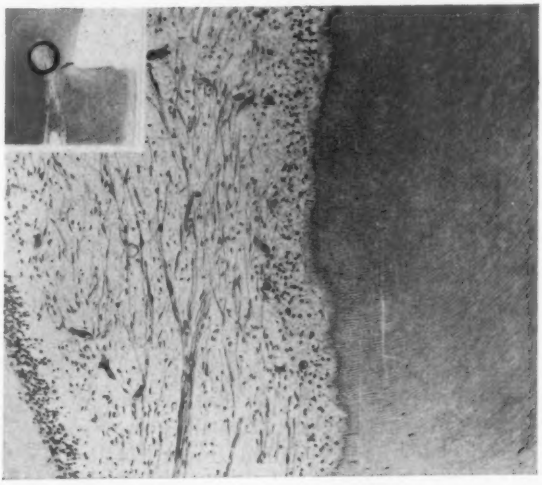

Fig. 3.—One-day "nonspray" specimen. Insert demonstrates loss of odontoblastic layer extending above the cut tubules. Enlargement shows very few displaced odontoblasts, loss of cellular detail in the odontoblastic layer, and lack of an inflammatory infiltrate. (Magnification: enlargement ×105, insert ×16; reduced ⅙.)

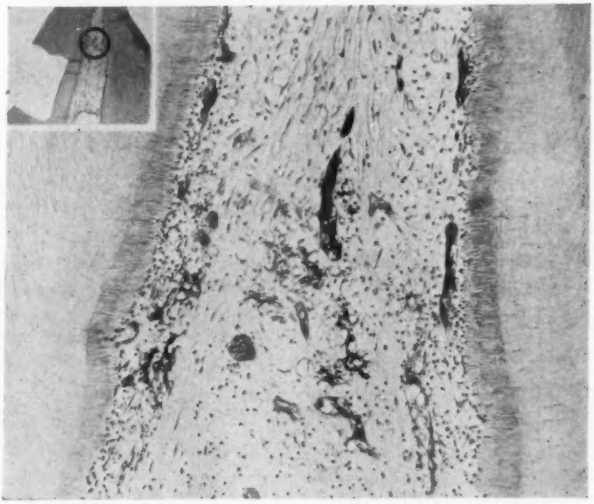

Fig. 4.—Twenty-day "nonspray" specimen. The inflammatory response is uniform on both sides of the pulp, as is the number of displaced odontoblasts. The insert permits the region of the pulp associated with the cavity preparation to be identified and shows the extent of the lesion above the cut tubules. (Magnification: enlargement ×105, insert ×10; reduced ⅙.)

cedure and to relate the phenomenon to inflammation itself. We postulated, as previously stated, that the phenomenon probably was the result of crowding of the pulpal tissues due to a build-up of intrapulpal pressure in a confined area by the edema, hyperemia, and exudation. The degree of inflammatory response determined the intensity of the odontoblastic displacement. By the end of the study period (thirty-six days) relatively few displaced odontoblasts were found, especially in specimens showing irregular dentine formation.

In February, 1957, Lefkowitz, Robinson, and Postle[12] found that cavities prepared by methods causing excessive heat were associated with displacement of odontoblasts. They state: "One significant finding is that cavity preparation which causes excessive heat is responsible for the aspiration of the odontoblasts into the dentinal tubules. The same methods, with a water coolant, will not cause aspiration of odontoblasts. The presence of aspirated odontoblasts in histologic sections indicates that excessive heat was generated in the preparation of the cavity, and is offered as a histologic diagnostic entity."

NEW DATA

We have collected twenty-four additional examples of displaced odontoblasts not associated with either dentinal tubules cut during cavity preparation or forceps injury but associated with an inflammatory response. These consisted of eleven cases of chronic pulpitis with intrapulpal abscesses due to former operative procedures, five cases of pulpitis resulting from carious exposures not complicated by operative procedures, one case of inflammation associated with the physiologic resorption of a deciduous tooth, two cases of inflammation associated with leukemic infiltrations, and five cases associated with inflammation in a pulp-amputation and capping study.* These specimens were processed in the same manner as previously described.[1] The following case reports were selected as examples of the variable circumstances under which displacement of odontoblasts and blood cells can be found.

CASE 1.—This case involved a maxillary first premolar of a 28-year-old woman.

History.—The patient developed symptoms of chronic pulpitis three weeks previously. At that time an MODL amalgam was replaced with a temporary filling. Symptoms persisted, leading to an acute pulpitis episode.

Microscopic Findings (Fig. 5).—Necrotic debris is seen in a pulpal horn narrowed by large deposits of irregular dentine. An abscess cavity marginated by immature granulation tissue extends from the pulpal horn to below the cementoenamel junction and across the pulp chamber to the opposite wall. Displaced odontoblasts are conspicuous in intact tubules far removed from the operative site but adjacent to an acute inflammatory reaction in the adjacent pulp tissues.

CASE 2.—The tooth involved was a maxillary left second premolar of a man whose age was not recorded.

History.—A carious exposure occurred beneath an old amalgam restoration. A temporary filling was placed, and the tooth was extracted twenty-four hours later.

*These specimens were part of a study conducted by Dr. Harold Berk while on assignment to the National Institutes of Health, Bethesda, Maryland.

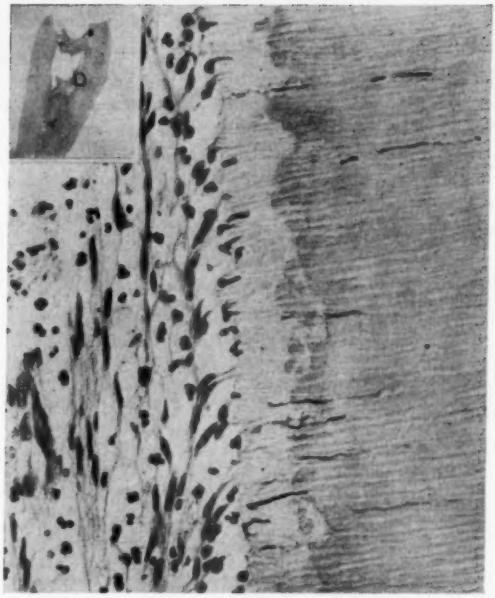

Fig. 5.—Maxillary first premolar of 28-year-old woman; chronic pulpitis. Insert reveals a large space partly resulting from loss of abscess contents and partly from shrinkage artifact. Enlargement reveals edematous changes in the odontoblastic layer, large numbers of leukocytes, and displaced odontoblasts. (Magnification: enlargement ×350, insert ×2.5; reduced ⅙.)

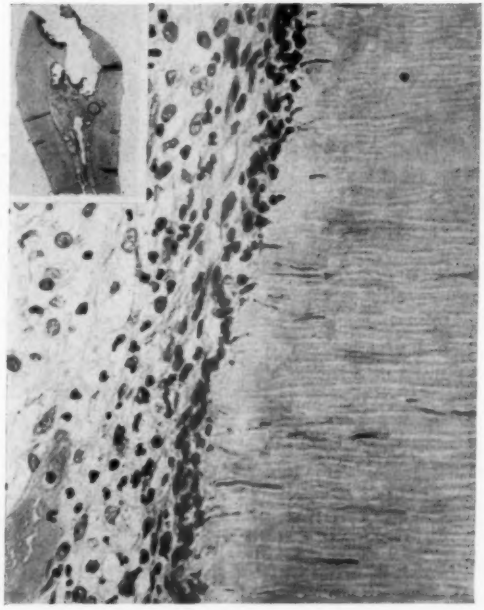

Fig. 6.—Maxillary second premolar of an adult male patient; a carious exposure. Note acute inflammatory reaction and displacement of cells. (Magnification: enlargement ×350, insert ×2.5; reduced ⅙.)

Microscopic Findings (Fig. 6).—The coronal pulp consists mostly of loose, edematous, chronically inflamed granulation tissue with many dilated, congested capillaries. Focal abscesses are present. Eosinophils and leukocytes are prominent. A perivascular exudate predominating in plasma cells is seen extending into the root canals. Displaced odontoblasts are found in intact dentinal tubules quite remote from the operative site but accompanied by an acute inflammatory response in the subjacent pulp tissues.

CASE 3.—The tooth was the mandibular right first premolar of an 8-year-old boy.

History.—A normal, noncarious tooth scheduled for removal for orthodontic reasons received an experimental pulpotomy two weeks prior to extraction.

Microscopic Findings (Fig. 7).—A developing barrier in response to the treatment consists predominantly of fibrous tissue running horizontally across the uppermost pulpal surface. Although the inflammatory response is limited, a wedge of inflammed tissue extends apically along the buccal wall. Displaced odontoblasts and leukocytes are conspicuous within intact dentinal tubules approximating the area.

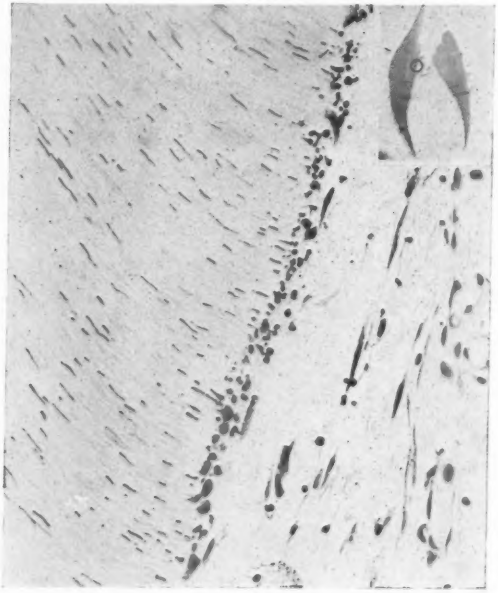

Fig. 7.—Mandibular first premolar from 8-year-old boy; experimental pulpotomy. Note prominent number of displaced cells. Segmented nuclei of leukocytes can be observed in the tubules as well as the long slender odontoblastic nuclei. (Magnification: enlargement ×280, insert ×2.5; reduced ⅙.)

CASE 4.—A deciduous mandibular cuspid in a 7-year-old boy was exfoliated normally without instrumental assistance. The patient had myelogenous leukemia.

Microscopic Findings (Fig. 8).—A noncarious tooth exhibiting advanced resorption of the root structure with prominent scalloping of the remaining root surface. Many of the resorption lacunae contain multinucleated osteoclasts (dentinoclasts). The pulp tissue is vital but reveals acute inflammation with a mixed cellular infiltrate consisting of leukemic cells and mature leukocytes and lymphocytes. Wherever leukocytes have infiltrated the odontoblastic layer, displacement of odontoblasts has occurred. In the center of the pulp is a focus of extramedullary myeloid hematopoietic tissue.

In addition, a study dealing with the autolytic changes in the pulps of normal human teeth revealed generalized, shallow displacement of odontoblasts. The severe vacuolization of autolysis approximated the displaced cells.

DISCUSSION

Orban[6] suggested that leukocytes in dentinal tubules represented a chemotactic response. However, this did not explain the presence of erythrocytes, whose motion is known to be passive,[13] or other fixed tissue cells, such as odontoblasts.

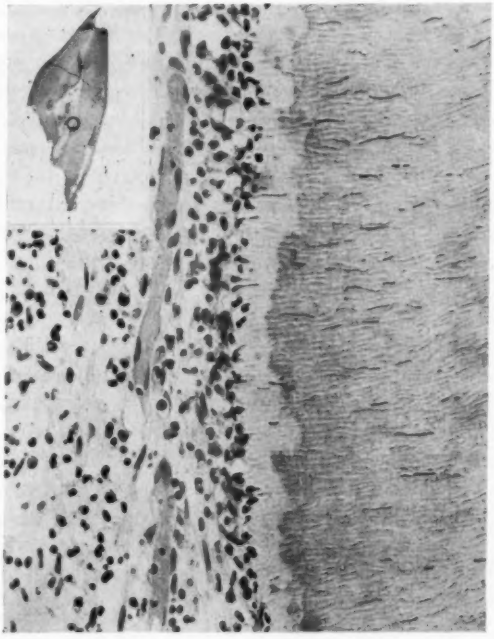

Fig. 8.—Mandibular deciduous cuspid from 7-year-old boy; normally exfoliated without instrumental assistance. Immature leukocytes can be distinguished in the infiltrate beneath the tubules containing displaced cells. (Magnification: enlargement ×280, insert ×4; reduced ⅙.)

Rushton[8] inferred that the leukocytes, like odontoblasts, might be sucked into the tubules on release of forceps pressure. This explanation would imply a generalized change in intrapulpal pressure and, consequently, might be expected to produce a more widespread displacement of pulpal cells than actually occurs. Kramer and McLean[3] did not relate the leukocytic infiltrations that were so prominent by the seventh postoperative day with the simultaneous displacement of odontoblasts. This was in contrast to Orban's[6] original association of an inflammatory response with displaced odontoblasts. Our grinding technique

produced enough pulpal injury to cause inflammation and associated odontoblastic displacement despite the presence of zinc oxide and eugenol. However, Kramer and McLean's technique apparently required the added insult of the self-curing acrylic resins to produce sufficient injury. This probably explains the lack of displacement in their control teeth filled with zinc oxide and eugenol.

We,[1] as well as James and Schour,[10] demonstrated that within one hour postoperatively odontoblasts appeared in the tubules of predentine in the presence of edematous changes. Although we noted a decreased number of displaced odontoblasts after thirty-six days, we did not observe the granular debris described by James and Schour at the end of two weeks, which the latter interpreted as fragmented remains of odontoblastic nuclei.

Lefkowitz and colleagues,[12] while they did not fully describe either their operative procedure or the type and speed of handpiece used, found under the conditions of their experiment that a coolant was adequate to prevent the degree of thermal damage necessary to induce odontoblastic displacement. As a result of their findings, these workers concluded that whenever odontoblastic displacement occurred it was indicative of thermally induced pulpal injury. They emphasized that this phenomenon was conclusively diagnostic of excessive heat generated during cavity preparation. Whereas we[1] recognized that the thermal injury may induce inflammatory changes which are associated with various types of cells in the dentinal tubules, we also realized that displacement could occur under a wide variety of circumstances.

While the presence of cells in tubules has become associated with negative pressures as a result of the introduction of the term "aspiration," it now becomes apparent that this phenomenon is the result of any circumstance which is associated with an increase in intrapulpal pressure. This is readily understood in the case of inflammatory changes. It is also conceivable that the odontoblastic displacement mechanically induced by forceps trauma during extraction is the result of a localized increase in intrapulpal pressure which may develop when the root dentine, which is slightly elastic, is subjected to high compression by the beaks of the forceps. All our evidence to date suggests that this is a pressure phenomenon.

SUMMARY AND CONCLUSIONS

This study was conducted to evaluate the literature and to contribute new data in an effort to help resolve the problem of cells displaced into dentinal tubules.

All the evidence seems to indicate that this phenomenon is generally the result of crowding of the pulpal tissues due to a build-up of intrapulpal pressure by an inflammatory response. Whenever edema, hyperemia, and exudation occur in proximity to the pulpal wall, odontoblasts and blood cells may be displaced into the dentinal tubules. This may occur regardless of the cause as evidenced by its association with operative trauma, carious exposures, physiologic exfoliation, leukemic infiltrations, and pulpotomies.

It is obvious that displacement (a term preferred to aspiration) of cells can occur under many different pathologic circumstances and should not be considered a histologic diagnostic criterion of excessive heat created during cavity preparation.

Generalized displacement can be seen in intact teeth undergoing autolytic changes.

Grateful acknowledgment is made to Alexander M. Schwollman for preparing the microscopic sections and to Robert R. Padgett for preparing the photomicrographs.

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ANEURYSMAL BONE CYSTS OF THE MANDIBLE

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*Surindar N. Bhaskar, Major, DC, USA***

In 1942 Jaffe and Lichtenstein[1] segregated the aneurysmal bone cyst from other solitary cystic lesions of bone, and later each gave his own detailed account of this clinicopathologic entity.[2, 3] A review of the literature shows, however, that lesions histologically identical to the so-called aneurysmal bone cyst have been described under a variety of names, among which were hemorrhagic osteomyelitis,[4] ossifying hematoma,[5] osteitis fibrosa cystica[6] and aneurysmal giant-cell tumor.[7] Dahlin and associates[8] reported twenty-six cases. From their study and from those of Jaffe[2] and Lichtenstein,[3] it would appear that the aneurysmal bone cyst is characterized by the following clinical and histologic features:

1. The patients are predominantly under 20 years of age.

2. A history of trauma is common.

3. Lesions have been reported in the vertebral column, long bones, clavicles, ribs, occipital bone, metacarpals, carpals, metatarsals, sacrum, innominate bone, and scapula. According to Jaffe,[2] 75 per cent occur in the vertebral column and long bones. Dahlin and associates[8] reported fifteen lesions in long bones and only five in the vertebrae.

4. Except for one case in which the occipital bone was involved,[8] lesions of the calvarium or facial bones have not been reported.

5. In long bones the shaft is the predominant, but not the exclusive, site of involvement. In flat bones the lesion occurs at the end of the bone or at some distance from the articulation.

6. Major complaints are pain accentuated by exercise, limitation of movement if the lesion is near a joint, swelling if the lesion is not deep, and neurological disturbances in some cases of vertebral involvement.

7. The roentgenologic features are characteristic. The lesion has a honeycomb or soap-bubble appearance. In the long bones it is well circumscribed and usually eccentric. The cortex may be destroyed, and new periosteal bone formation may be apparent. In smaller bones or flat bones the entire width of the bones is affected to produce a symmetrical deformity.

8. On surgical exploration the lesion exhibits "welling of blood" or excessive bleeding.

9. Grossly, the lesion has a spongy appearance with blood-filled cavities.

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10. Microscopically, it consists of numerous blood-filled, cavernous spaces lined by young connective tissue but devoid of endothelial lining, elastic, or muscular elements. The connective tissue walls contain hemosiderin and varying numbers of giant cells. The blood spaces do not show evidence of thrombosis. Bone formation may be seen in the connective tissue.

11. The lesion is benign and local curettage is curative.

Aneurysmal bone cyst of the mandible or the facial bones has never been reported. It should not be mistaken for and has nothing in common with the arteriovenous fistula[9] or aberrant blood vessels or the so-called latent bone cavity.[10] The following cases in which the site of the lesion is the mandible are of particular interest.

Fig. 1.

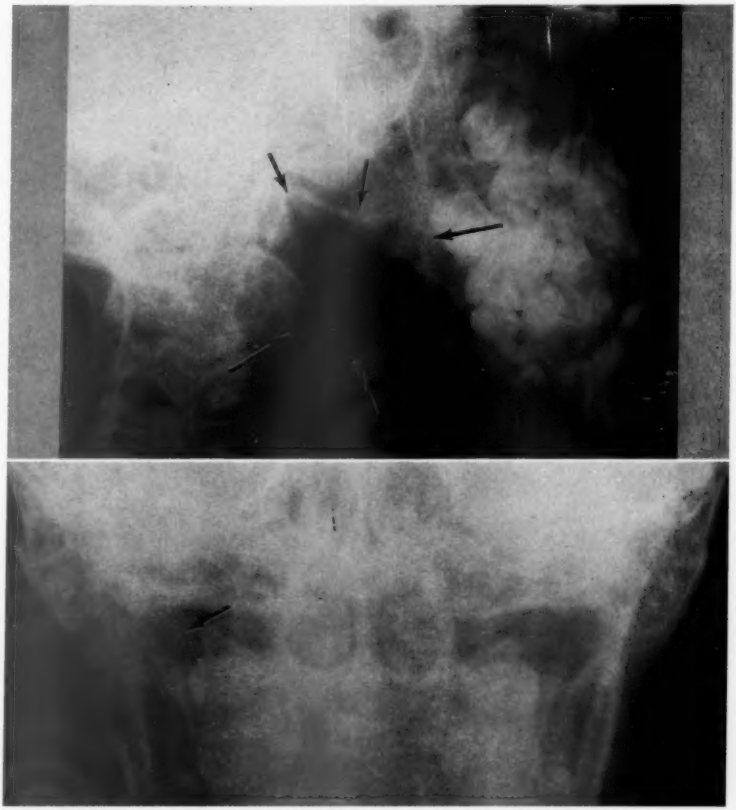


Fig. 2.

Fig. 1.—Case 1. The cystic lesion involves portions of the coronoid process and ramus. (AFIP Accession No. 743424.)
Fig. 2.—Case 1. The expansive cystic lesion on the right side. The cortex is destroyed.

CASE 1.—

Clinical History.—The parents of an 11-year-old girl noted a swelling over the right side of her mandible. It increased in size and became tender during the two months before examination, when a firm, tender, bony mass the size of a "hen's egg" was palpable

Fig. 3.

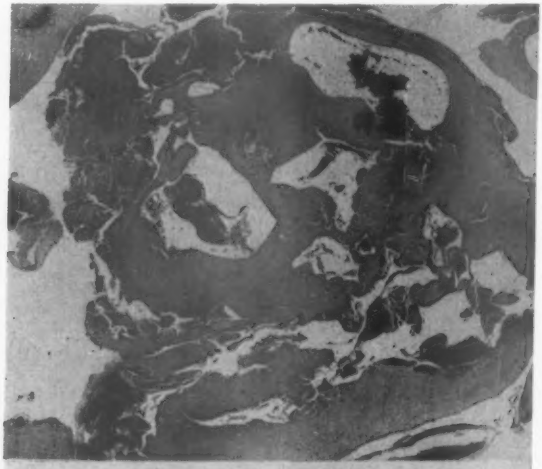


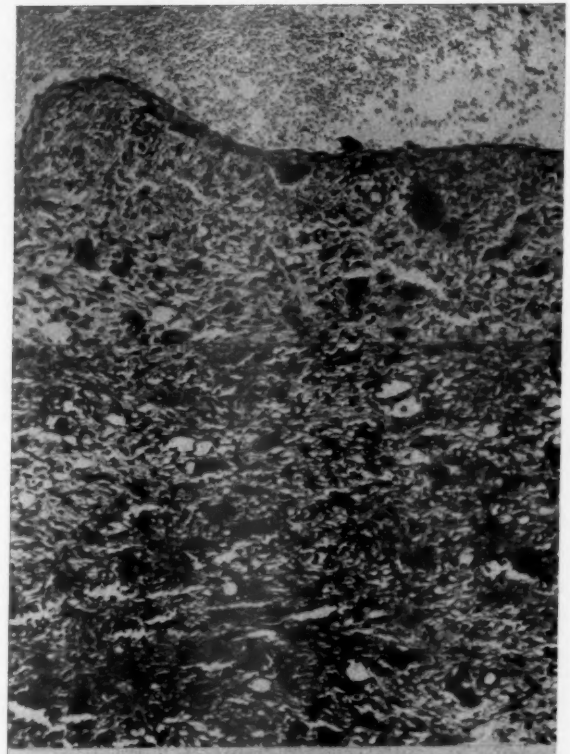
Fig. 4.

Fig. 3.—Case 1. Aneurysmal bone cyst, showing numerous cavernous blood spaces. (AFIP Negative 57-1046.) (Hematoxylin and eosin stain. Magnification, ×12; reduced ⅙.)

Fig. 4.—Case 1. Wall of blood channel, showing numerous giant cells. (Hematoxylin and eosin stain. Magnification, ×56; reduced ⅙.)

anterior to the right ear. A lateral roentgenogram showed a large unilocular cystic lesion involving the ramus and extending into the condylar and coronoid processes (Fig. 1) but sparing the temporomandibular joint. In the anteroposterior roentgenogram it was seen that a large balloon-shaped deformity of the mandibular ramus had destroyed the cortical

Fig. 5.



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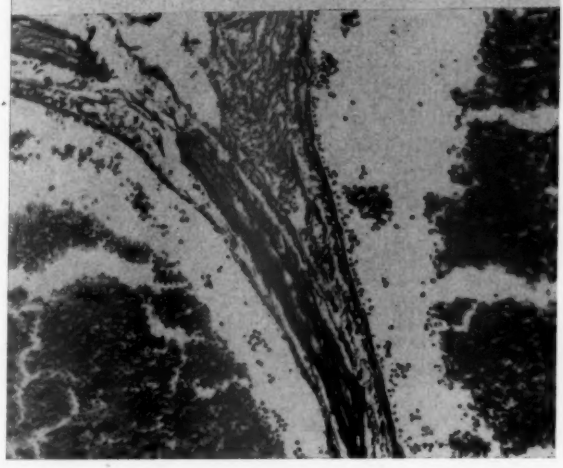


Fig. 5.—Case 1. Wall of a large blood channel. Note the cellular fibroblastic stroma, capillaries, and giant cells. (AFIP Negatives 57-1607 and 57-1614.) (Hematoxylin and eosin stain. Magnification, ×84; reduced ⅓.)

Fig. 6.—Case 1. A portion of the aneurysmal bone cyst to show the relationship of the reticular fibers to the blood channels. (AFIP Negative 57-1611.) (Gomori's reticulum stain. Magnification, ×200; reduced ⅓.)

Fig. 7.

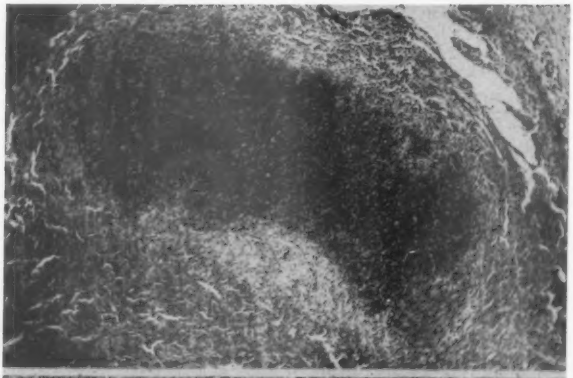
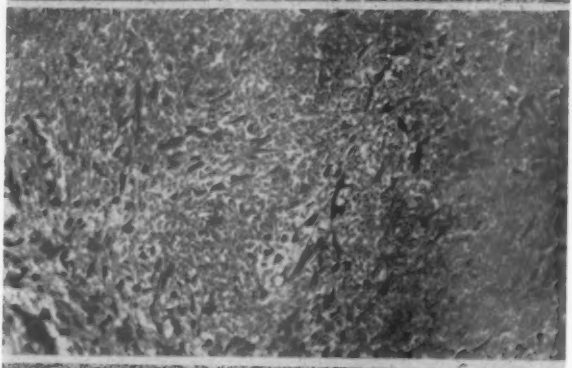


Fig. 8.



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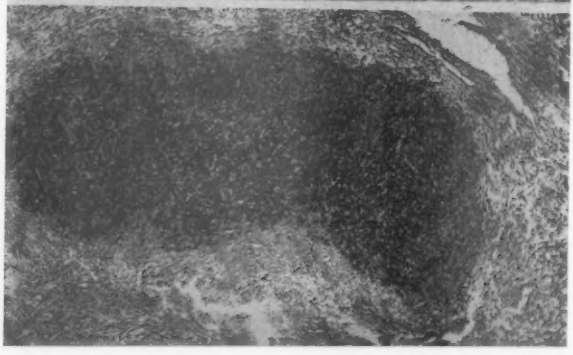

Fig. 7.—Case 1. A focus of hemorrhage in the stroma. (AFIP Negative 57-1604.) (Hematoxylin and eosin stain. Magnification, ×42; reduced ⅕.)

Fig. 8.—Case 1. High-power photomicrograph of an area shown in Fig. 7. Note the fibroblastic invasion of blood pool. (AFIP Negative 57-1617.) (Hematoxylin and eosin stain. Magnification, ×60; reduced ⅕.)

Fig. 9.—Case 1. Gomori's reticulum stain of the area seen in Fig. 7. (AFIP Negative 57-1609.) (Magnification ×42; reduced ⅕.)

plate (Fig. 2). Although the lesion had expanded the bone both laterally and medially, the deformity was more pronounced on the lateral surface.

At surgical exploration, the cortex of the outer surface of the ramus was thinned and showed irregular elevations. The bone defect contained blood-filled, yellowish gray, spongy tissue. The cavity was curetted and filled with bone chips. Recovery was uneventful, and one year and three months after the operation there was no evidence of recurrence.

The specimen consisted of multiple fragments of soft tissue with focal areas of hemorrhage and multiple clefts and cystic spaces which were from 1 to 5 mm. in diameter.

Fig. 10.

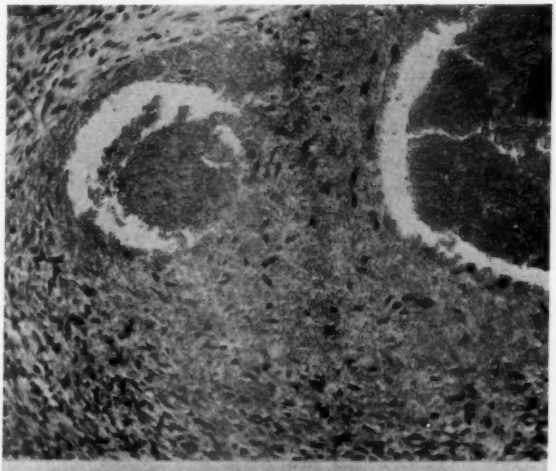

Fig. 11.

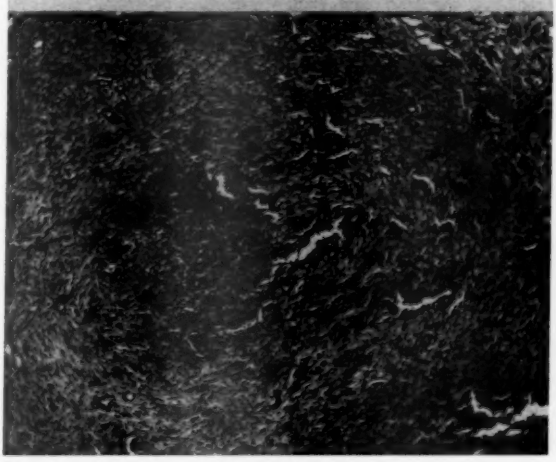

Fig. 10.—Case 1. Organization in part of a blood channel. (AFIP Negative 57-1060.) (Hematoxylin and eosin stain. Magnification, ×140; reduced ⅕.)

Fig. 11.—Case 1. An area in the stroma of the aneurysmal bone cyst, showing a focus of red cells, giant cells and fibroblasts. (AFIP Negative 57-1612.) (Hematoxylin and eosin stain. Magnification, ×84; reduced ⅕.)

Histologic Findings.—The most striking microscopic feature of the lesion was the presence of numerous large and small blood-filled cavernous spaces (Fig. 3). Most of these spaces were lined by highly cellular, young connective tissue, some parts of which contained many giant cells and erythrocytes and considerable hemosiderin (Figs. 4 and 5). The fibroblasts forming the innermost layer of the lining of the blood spaces were often flattened and fusiform and superficially resembled endothelial cells. Special stains failed to demonstrate any elastic tissue in the walls of the blood spaces, while reticulum stains revealed that the reticular fibers extended to their very edge (Fig. 6).

The stromal tissue of the lesion consisted of young fibroblasts, unevenly distributed foci of multinucleated giant cells (Fig. 5) and extra- and intracellular hemosiderin. Sparsely dispersed throughout the lesion, both in the stromal tissue and immediately around the blood pools, new bone formation could be seen. In general, the stromal tissue was identical to that of the giant-cell reparative granuloma (Fig. 5).

In the stroma, circumscribed foci of hemorrhage were present and these were infiltrated by varying numbers of fibroblasts (Figs. 7 and 8). Special stains revealed a network of argyrophilic fibers in these foci (Fig. 9). Fibroblastic infiltration and the reticular network were also seen in some of the blood channels (Fig. 10). Some places revealed small foci of hemorrhage, fibroblasts, hemosiderin, and giant cells which were surrounded by highly cellular fibroblastic stroma (Fig. 11).

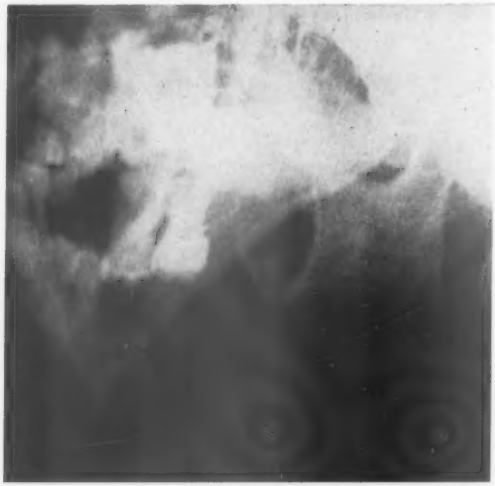

Fig. 12.—Case 2. Note the large radiolucent area in the mandible. (AFIP Accession No. 736869.)

CASE 2.—

Clinical History.—A 59-year-old Negro woman noticed a "tumor" at the site of an extracted mandibular second molar. In five weeks the "tumor" grew until it extended to both the buccal and the lingual sides of the ridge. There were accompanying swelling and deformity of the mandible, but the lesion was painless. The surface of the "tumor" was necrotic, and the underlying mandible had enlarged to the size of a walnut. Roentgenograms showed a large radiolucent area in the molar region (Fig. 12). The general health of the patient was good, and other clinical and laboratory findings were negative.

Fig. 13. Fig. 14.

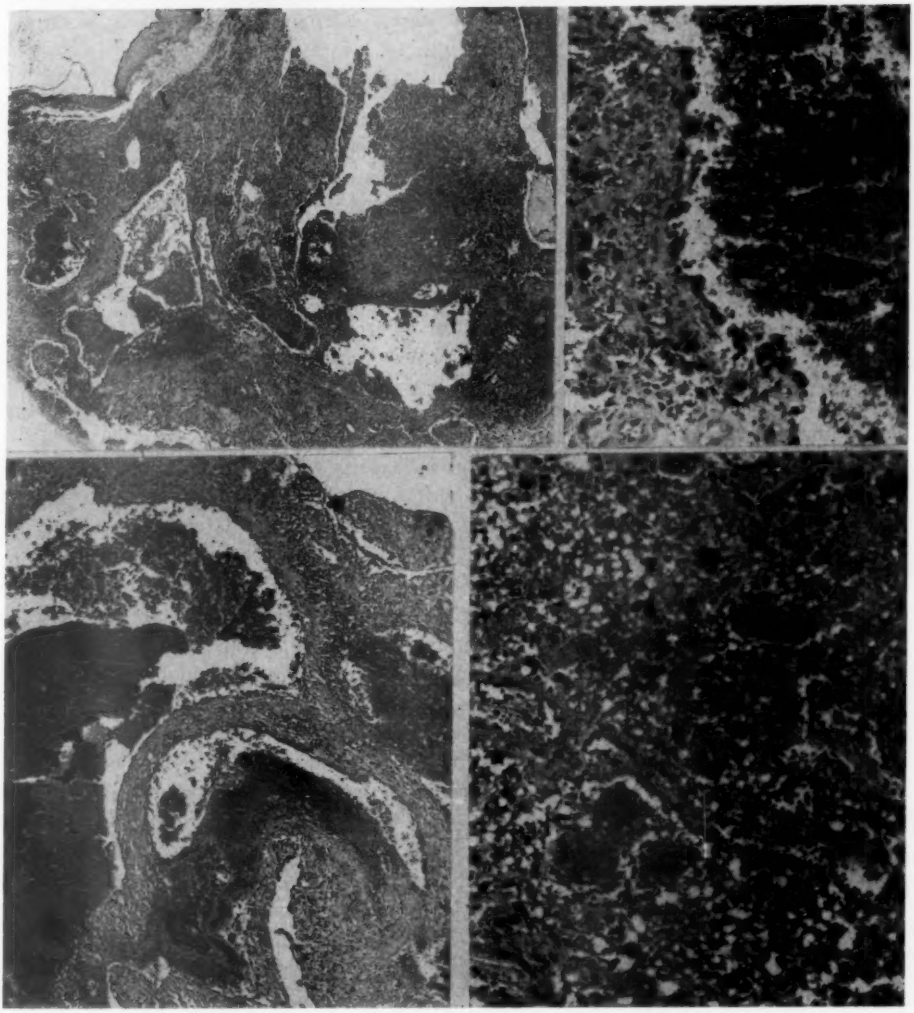

Fig. 15. Fig. 16.

Fig. 13.—Case 2. Aneurysmal bone cyst. Portion of the lesion involving the gingival tissue. Note the covering stratifed squamous epithelium. (AFIP Negative 57-7492.) (Hematoxylin and eosin stain. Magnification, ×15; reduced ⅙.)

Fig. 14.—Case 2. The tissue lining the blood-containing spaces is characteristic of the aneurysmal bone cyst. (AFIP Negative 57-7487.) (Hematoxylin and eosin stain. Magnification, ×130; reduced ⅙.)

Fig. 15.—Case 2. Note large blood-containing spaces. (AFIP Negative 57-5676.) (Hematoxylin and eosin stain. Magnification, ×50; reduced ⅙.)

Fig. 16.—Case 2. Stroma, showing features of giant-cell reparative granuloma. (AFIP Negative 57-5681.) (Hematoxylin and eosin stain. Magnification, ×200; reduced ⅙.)

The surgeon extracted the teeth remaining at the site and removed the gingival mass. It was described as mushroom-shaped and extending deep into the mandible. The mass in the bone was easily removed; the area was curetted; iodoform gauze was placed in the defect and held in place with a suture. Healing was uneventful.

Grossly, the specimen consisted of multiple fragments of tissue which appeared hemorrhagic.

Microscopic Findings.—The mircoscopic features of this lesion were identical to those described in Case 1 (Figs. 13 to 16). Numerous blood-filled spaces lined by giant cells were a prominent feature. The stroma contained fibroblasts, giant cells, and numerous capillaries. The gingival part of the lesion was covered by stratified squamous epithelium and consisted of tissue identical to that of the aneurysmal bone cyst (Fig. 13). This case was unusual in that the so-called aneurysmal bone cyst involved not only the bone but extended also into the soft tissue.

DISCUSSION

Whereas the histologic features of the aneurysmal bone cyst are characteristic, its histogenesis is not well established. Ewing[7] considered this lesion an aneurysmal variant of the benign giant-cell tumor and referred to it as such. He believed that the prominent vascular feature was a result of free communication with large blood vessels. Lichtenstein[3] has suggested, however, that the aneurysmal bone cyst probably arises as a result of a circulatory disturbance, such as thrombosis of a large vein or an anomalous arteriovenous communication with resultant dilatation and congestion of the vascular bed. Subsequently, resorption of the spongy bone and the cortex occurs. The bone thus resorbed is replaced by fibrous connective tissue, osteoid, and new bone. In his opinion, the giant cells are a result of bone resorption and reconstruction, and the numerous blood channels seen in the aneurysmal bone cyst are dilated capillaries and venules. In our study, however, special stains failed to reveal elastic or muscular tissue in the wall of any of these blood channels. They are devoid of endothelial lining, and in reticulum stains the argyrophilic fibers can be traced to the walls of the blood pools (Fig. 6). It is difficult, therefore, to accept these spaces as pre-existing blood vessels which have undergone dilatation. Consequently, the hypothesis that the aneurysmal bone cyst follows a venous thrombosis and represents a vascular bed which has merely become congested seems untenable. In our cases some of the blood channels showed evidence of thrombosis. In the areas of hemorrhage within the stroma the sequence of events was believed to be as follows: In the beginning, the foci of hemorrhage contained a few fibroblasts and abundant argyrophilic fibers. Later the fibroblasts increased, hemosiderin was taken up by phagocytes, giant cells appeared, reticular fibers became fewer, and collagen fibers progressively increased. In some areas foam cells could be seen. Finally, foci of hemosiderin, fibroblasts, giant cells, and isolated erythrocytes remained (Fig. 11). These changes suggest than in this lesion at least some of the stromal features are a result of organization of hemorrhage. This is seen not only in the hemorrhagic foci of the stroma but also in some of the blood spaces, as shown in Fig. 10. Thus,

the most prominent components of the stroma—namely, fibroblasts, hemosiderin, and giant cells—are interpreted as a peculiar reparative response to hemorrhage. We have already observed that, on the basis of their morphologic features, the blood-containing spaces cannot be considered pre-existing blood vessels of any type. It would appear, rather, that they represent unorganized or canalized portions of a hematoma. Because uncoagulated blood is seen in these spaces and because the lesion "wells up" with blood at operation, it is logical to assume that there is a flow of blood through them. The lesion, therefore, might represent a "false" aneurysm of a blood vessel in which part of the hematoma is replaced by peculiar connective tissue complex, while in part a slow flow of blood is maintained. Such a condition undoubtedly would be rare, and this indeed is the clinical record.

Although the giant cells form a constant and prominent part of the aneurysmal bone cyst, this lesion is undoubtedly distinct from the giant-cell tumor. Such a conclusion is based on the observation that the two differ not only in their microscopic structure but also in age incidence, clinical features, roentgenologic appearance, and prognosis (Table I). It appears, however,

TABLE I. DIFFERENTIAL FEATURES OF GIANT-CELL TUMOR, ANEURYSMAL BONE CYST, AND GIANT-CELL REPARATIVE GRANULOMA

GIANT-CELL TUMOR	ANEURYSMAL BONE CYST	GIANT-CELL REPARATIVE GRANULOMA
1. Occurs predominantly in long bone, rare in vertebrae	Occurs frequently in vertebrae	Mainly in mandible and maxilla
2. Predominantly epiphyseal in location	When in long bones, metaphyseal in location	Central and peripheral
3. Rare in patients under 20 years of age	Predominantly in patients less than 20 years of age	Predominantly in patients less than 20 years of age
4. Marked tendency to recur—some metastasize	Does not recur—local curettage curative	Local curettage curative
5. Histologically, a more or less even distribution of giant cells; collagenization and bone formation rare	Histologically, unevenly distributed giant cells, hemosiderin, blood-containing spaces, blood vessels, and fibroblasts, collagenization and bone formation	Histologically, unevenly distributed giant cells, hemosiderin, blood vessels, fibroblasts, collagenization, and bone formation

that the aneurysmal bone cyst does resemble the central giant-cell reparative granuloma seen in the jaws. Both lesions occur most frequently in patients less than 20 years of age, are endosteal in location, enlarge the affected bone, and are usually cured by local curettage. In both lesions giant cells are unevenly distributed, and hemosiderin pigment, osteoid, bone, and areas of collagenization may be seen. The only difference between them is the presence of numerous blood spaces in the aneurysmal bone cyst. It may be that the giant-cell reparative granuloma represents an organized hematoma in which the damaged blood vessels fail to retain a circulatory connection with the lesion.

SUMMARY AND CONCLUSIONS

The present report deals with two cases of aneurysmal bone cyst of the mandible. The lesions were identical to those described in other parts of the skeleton. These cases were unique in that they represent the second and third such cysts known to have occurred in the craniofacial skeleton and the only two in any of the facial bones, including the mandible.

It is postulated that the aneurysmal bone cyst, though having nothing in common with the giant-cell tumor of bone, resembles the central giant-cell reparative granuloma of the jaws in many ways. The only difference between the aneurysmal bone cyst and the giant-cell reparative granuloma is the presence of blood-containing spaces in the former. Regarding their histogenesis, it is suggested that both lesions represent overzealous attempts of connective tissue to replace a hematoma in the bone marrow. If the hematoma maintains a circulatory connection with the damaged vessel an "aneurysmal bone cyst" results, while if such a connection is obliterated the ensuing lesion is a giant-cell reparative granuloma.

Case 1, AFIP Accession 743424, was submitted for consultation to the Armed Forces Institute of Pathology by Dr. Roscoe B. Anderson, pathologist at the Riverside Community Hospital, Riverside, California, and Case 2, AFIP Accession 736869, by Dr. James T. McClellan, pathologist at Lexington Clinic, Lexington, Kentucky. These contributions are gratefully acknowledged. The histologic material for this study was prepared by Miss Eliza B. Buddo of the D & O Branch, Armed Forces Institute of Pathology.

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ENDODONTICS

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A COMPARATIVE STUDY OF THREE ROOT CANAL SEALING AGENTS

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THE available textbooks on endodontics agree that the root canal should be thoroughly sealed after it has been freed of infection.[1, 3, 9] They present many methods of achieving this goal, and an endless variety of substances have been suggested for obturation of the canal. In spite of this, obturation has remained one of the weakest links in successful endodontic treatment.

Dow and Ingle[2] have stated that, in a controlled study, 50 per cent of the endodontic failures could be associated with poor root canal fillings. They have illustrated the inadequacy of determining complete obturation of the canal from a radiograph. They have also shown, by means of autoradiograph, the penetration of a radioactive solution (I^{131}) down the incompletely obturated canal.

Rickert and Dixon[8] have stated that fluid circulation into the interstices of a poorly filled root canal will invariably lead to periapical inflammation. Today we have a more complete explanation of this mechanism, as presented by Menken[6] in his new book entitled *Biochemical Mechanisms in Inflammation.* In the presence of inflammation, necrosin is liberated. This substance is also capable of producing areas of rarefaction in bone.

Most of us have at some time experienced failure in what we thought was a properly treated root canal which, radiographically, showed a thoroughly condensed filling. This failure could be attributed to resorption of the root canal sealing agent, from the apical third of the canal, with the resultant radiographic evidence of periapical pathology. One such case is illustrated in Fig. 1.

It is this type of failure that spurs one to seek a more reliable sealing agent for obturation of the root canal and to establish criteria for an ideal root canal sealer. Grossman has listed the following prerequisites for such a sealer:

1. It should be easily introduced into a root canal.
2. It should preferably be a semisolid upon insertion and become solid afterward.

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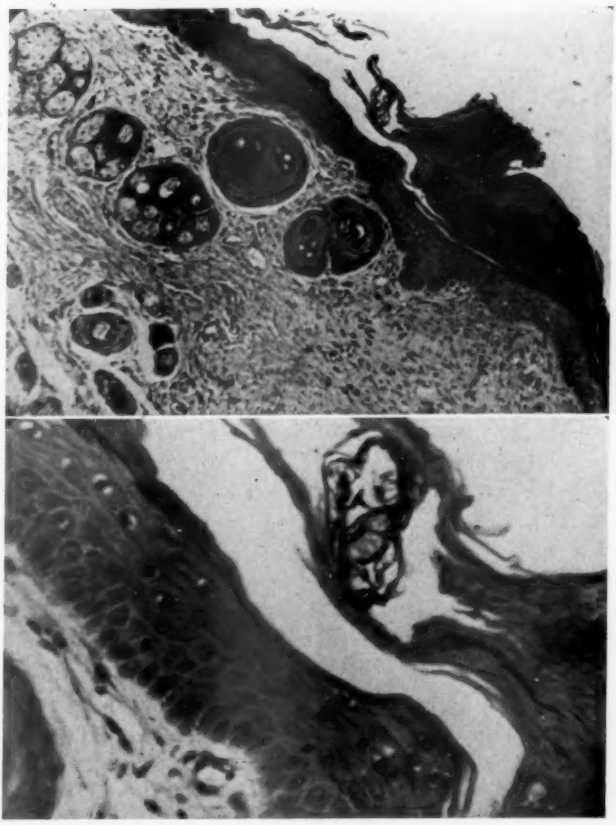
3. It should seal the canal laterally as well as apically.
4. It should not shrink after being inserted.
5. It should be impervious to moisture.
6. It should be bacteriostatic or at least not encourage bacterial growth.
7. It should be radiopaque.
8. It should not stain tooth structure.

Fig. 1.—*A*, Lateral incisor tooth requiring root canal therapy. *B*, Tooth treated and canal filled. Root canal paste was forced to the apex in the hope of filling that portion of the canal that was twisted.

9. It should not irritate periapical tissues.
10. It should be sterile, or it should be easily and quickly sterilized immediately before insertion.
11. It should be easily removed from the canal, should removal become necessary.

A.



B.

Fig. 2.—Five-week wound.
A, Appearance of hair follicles and cutaneous epithelium in tissue taken from the abdominal wall of the rabbit. This is a section from a control pocket to show normal healing.
B, High-power view of tissue showing regular formation of epithelial layer, normal appearance of epithelial layer, and normal appearance of connective tissue cells.

Perhaps an additional prerequisite can be added to this list. This would be that the sealing agent have sufficient tensile strength to retain either a silver wire or a guttapercha point, so that it will not be disturbed during future restorative procedures.

Since paste materials alone are difficult to control during obturating procedures, and either silver or gutta-percha points are generally used in conjunction with a sealing agent, studies were designed to test three of the presently available root canal sealing agents alone and in combination with metal points and/or gutta-percha. The studies include tissue tolerance testing, microbial inhibition, tensile strength, permeability, and clinical investigation.

The following root canal sealers were investigated:

1. *Kerr sealer:*

   Liquid. Oil of clove, 78.0 per cent; Canada balsam, 22.0 per cent.

   Powder. Zinc oxide, 41.2 per cent; precipitated silver, 30.0 per cent; white rosin, 16.0 per cent; thymol iodide, 12.8 per cent.

2. *The New Grossman sealer:*

   Liquid. Eugenol.

   Powder. Zinc oxide, 40.0 per cent; Stabellite rosin, 30.0 per cent; bismuth subnitrate, 15.0 per cent; barium sulfate, 15.0 per cent.

3. *Diaket, a new polyketone compound (with 0.5 per cent dihydroxydichlorodiphenylmethane):*

   $R\text{-}CO\text{-}CH_2 - R + MeO$
   Me = Bivalent metal
   R = An organic substance

TISSUE TOLERANCE STUDIES

The object of these studies was to observe the reactions and to measure the weight loss, if any, of specimens of the various root canal sealers implanted in the soft tissues of the rabbit.

Procedures.—Four animals, to date, were used in this study. Following induction of anesthesia by ether inhalation, the abdominal wall of each animal was shaved with an electric clipper. A one-inch incision was made through the skin to the muscle layer, and a pocket was created by blunt dissection.

The specimen of root canal sealer was previously prepared according to directions of the manufacturer and was weighed on an analytical balance. This specimen was then placed in the created pocket, and the skin was closed with interrupted black silk sutures. The control wounds were also made, as described.

In animal 1 the following sealers were placed, in individually prepared skin pockets: (1) Diaket, (2) Kerr sealer, (3) zinc oxide-eugenol mix, and (4) control. In animal 2, Diaket, Kerr sealer, and a control were placed in individual skin pockets. In animal 3, Diaket and Kerr sealer were placed in individual pockets. This animal was also subjected to a control wound pocket. Animal 4 had the new Grossman sealer and Diaket implanted in individual skin pockets and in a control pocket as well.

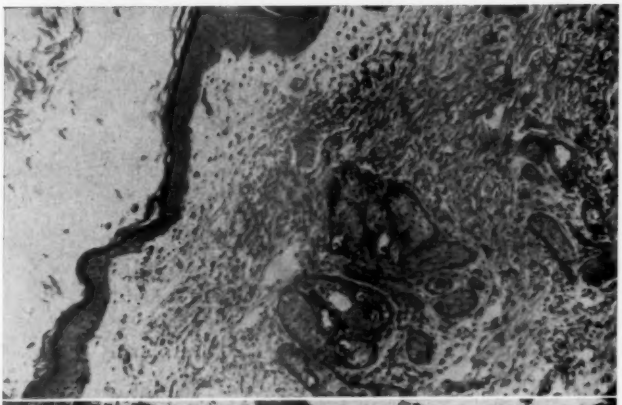


Fig. 3.

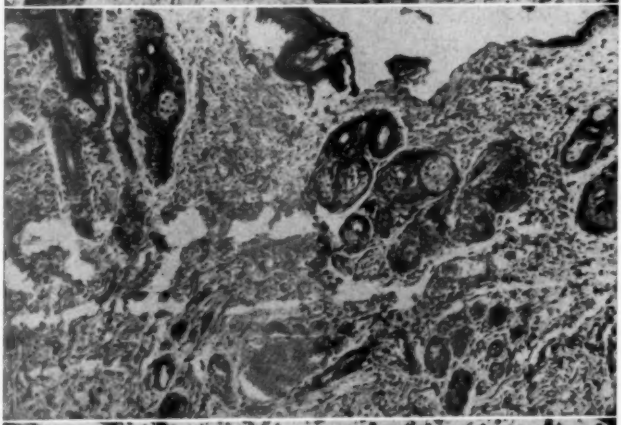


Fig. 4.

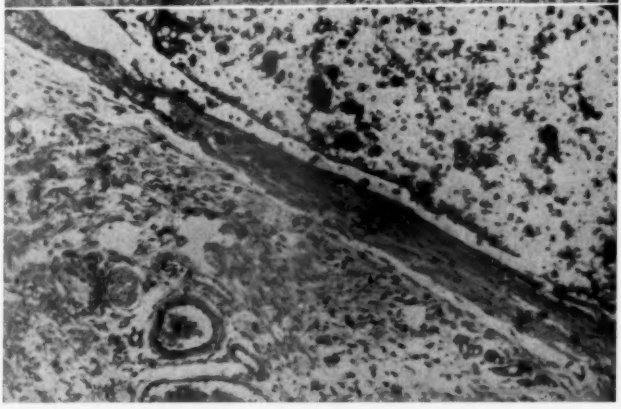


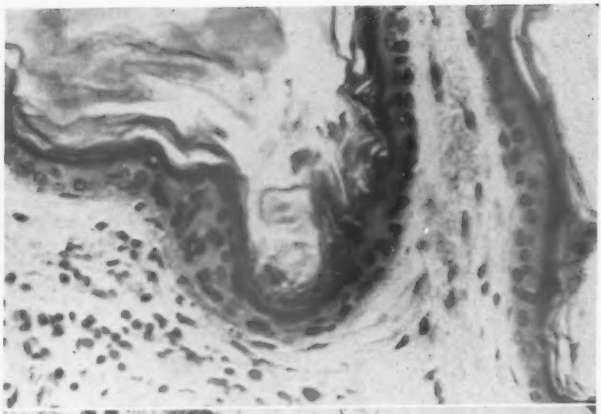
Fig. 5.

Fig. 3.—Appearance of hair follicles and cutaneous epithelium of rabbit tissue that was in contact with the new Grossman sealer for two weeks. There seems to be only slight inflammation.

Fig. 4.—Appearance of rabbit tissue (from abdominal wall) after two weeks' exposure to Diaket. Healing has progressed to a point paralleling the six-week Kerr sample.

Fig. 5.—Appearance of cutaneous tissue that had been in direct contact with Kerr sealer for five weeks. Note debris and silver particles, as well as moderate inflammation.

Fig. 6.



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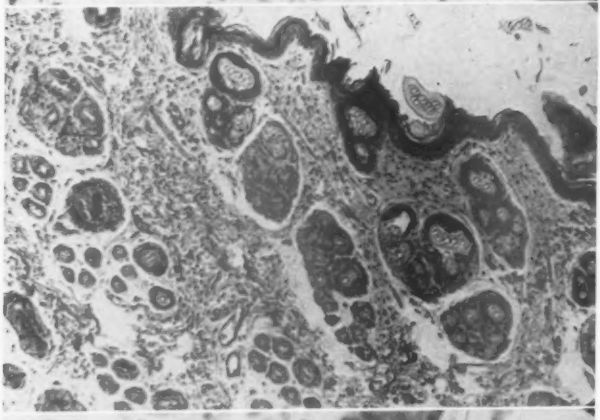


Fig. 8.

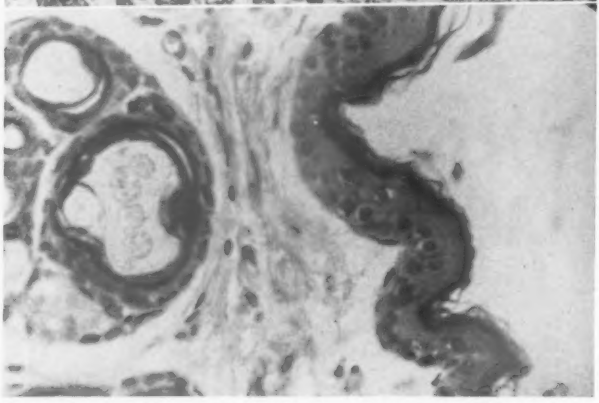


Fig. 6.—Appearance of cutaneous epithelium in n section of rabbit tissue exposed to Kerr sealer for five weeks. Note the increased cuticle layers and moderate round-cell infiltration.

Fig. 7.—Appearance of rabbit tissue showing hair follicles and cutaneous epithelium after being exposed to Kerr sealer for six weeks. It appears similar to the two-week specimen of Diaket-exposed tissue at low-power study.

Fig. 8.—Appearance of hair follicles and cutaneous epithelium in a section of rabbit tissue exposed to Diaket for six weeks. Note the regularity of the cellular structure and the lack of inflammation.

Findings.—The specimens were removed at varying intervals and weighed; the surrounding tissue was prepared and sectioned for microscopic study. Animal 1 was observed for a period of six weeks, animal 2 for a period of five weeks, animal 3 for a period of four weeks, and animal 4 for a period of two weeks.

Fig. 9.

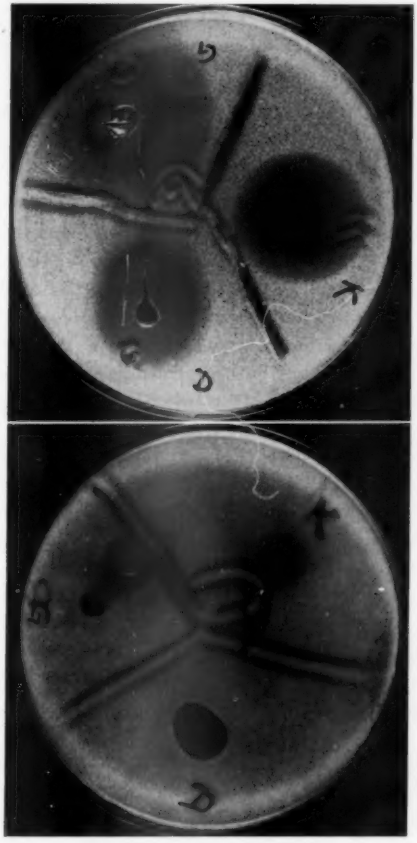


Fig. 10.

Fig. 9.—Note zones of inhibition of growth when Kerr sealer (*K*), Grossman sealer (*G*), and Diaket (*D*) are placed on a Petri plate seeded with the test organism, *Micrococcus citreus*, on nutrient agar.

Fig. 10.—Note zones of inhibition of growth when Kerr sealer (*K*), Grossman sealer (*G*), and Diaket (*D*) are placed on a Petri plate seeded with the test organism, *Staphylococcus albus*, on nutrient agar.

The zinc oxide–eugenol specimen was completely absorbed in the six-week interval. The other specimens, as evidenced by complete encapsulation, seemed to be equally well tolerated by the rabbit tissue. There was no apparent weight loss in the Kerr sealer samples tested, and no weight loss was noted

in the Diaket samples examined. There was a weight loss of 0.03 gram in the new Grossman sealer but, for all practical purposes, this loss is insignificant, since this minute amount could easily be accounted for in the manipulation of the tissues or specimen.

As is shown in the accompanying microscopic sections, the longer the specimens were permitted to remain embedded in the animal tissue, the heavier was the capsule and the more regular was the connective tissue.

MICROBIAL INHIBITION STUDIES

Since one of the prerequisites of an ideal root canal sealing agent is that it should inhibit bacterial growth, or at least not permit bacteria to grow, it was decided to test the three named agents for this quality.

Method.—The sealers were freshly prepared, again following the directions of the manufacturer. One drop of each sealer was placed in a seeded Petri plate. Brain-heart infusion agar and trypticase-soybean agar were used for all of the bacteria listed with the exception of the yeast organisms, which were grown on Sabouraud's agar. For each 10 c.c. of media, 0.2 c.c. of a twenty-four-hour suspension of the test organism was used. Each plate was incubated at 37° C. for a period of forty-eight hours before readings were taken.

The following organisms were studied:

1. An unidentified hemolytic streptococcus
2. A nonhemolytic streptococcus
3. *Streptococcus viridans*
4. Diphtheroids
5. A penicillin-resistant *Staphylococcus aureus*
6. *Escherichia coli*
7. *Staphylococcus albus*
8. *Staphylococcus aureus*
9. *Micrococcus citreus*
10. An unidentified yeast.

TABLE I

TEST ORGANISMS	MEDIUM					
	B.H.I.*	T.S.A.†	B.H.I.	T.S.A.	B.H.I.	T.S.A.
Hemolytic streptococcus	6.0	6.0	4.5	3.0	1.5	2.0
Nonhemolytic streptococcus	2.0	5.0	3.0	4.0	1.0	2.0
Staphylococcus viridans	5.0	5.0	3.5	3.0	3.0	2.0
Diphtheroids	4.0	10.0	6.0	5.0	2.0	2.0
Staphylococcus aureus (resistant)	1.5	3.0	2.0	3.0	2.0	1.5
Escherichia coli	4.0	10.0	2.5	3.0	2.0	3.0
Staphylococcus albus	3.0	4.0	3.0	2.0	1.5	1.5
Staphylococcus aureus	4.0	4.0	5.0	2.0	3.0	2.0
Micrococcus citreus	10.0	6.0	8.0	5.0	6.0	4.5
Yeast on Sabouraud's agar	4.0		7.0		4.5	
Sealer	Diaket		Grossman		Kerr	

Measurement recorded in millimeters of growth-free zone from edge of sample.
*B.H.I. = Brain-heart infusion agar.
†T.S.A. = Trypticase-soybean agar.

Findings.—Since there was some variation in the shape of the sealing agents when they dropped from the spatula and solidified on the agar surface, measurements were taken from the outer edge of the specimen to the beginning of colony growth. All of the substances tested showed some degree of microbial inhibition on all of the organisms tested. (See Table I for exact measurements.)

TENSILE STRENGTH STUDIES

Following endodontic treatment, a tooth occasionally may require a jacket crown to restore it to function. During this procedure it is not uncommon to find that the root canal point (or points), is unsuccessful. This is particularly true when one must resort to a post and crown.

In order to eliminate this possible danger of contamination, a root canal sealing agent which would hold a silver or gutta-percha point so securely that it would not be disturbed during these procedures would be desirable. With this in mind, the three root canal sealers were tested for their ability to retain wires which had been sealed both in extracted teeth and in specially prepared metal tubes.

POUNDS OF PULL REQUIRED TO REMOVE SEALED WIRE FROM METAL TUBE

		10 mm	15 mm	20 mm
DIAKET	.025	40.5 lb.	52.5 lb.	66.0 lb.
	.030	41.5 lb.	62.5 lb.	81.0 lb.
	.040	47.0 lb.	77.0 lb.	99.5 lb.

		10 mm	15 mm	20 mm
GROSSMAN	.025	14.5 lb.	42.5 lb.	47.0 lb.
	.030	22.0 lb.	44.5 lb.	69.0 lb.
	.040	41.5 lb.	57.0 lb.	76.5 lb.

		10 mm	15 mm	20 mm
KERR	.025	20.5 lb.	33.0 lb.	58.0 lb.
	.030	26.5 lb.	37.0 lb.	59.0 lb.
	.040	37.0 lb.	41.0 lb.	74.0 lb.

Table II.

Procedure.—In some of the preliminary tests with both the Diaket and the new Grossman sealer, it was noted that when the silver point was sealed in the extracted tooth and in the metal cylinder, it was held so securely that, upon attempted removal, the point itself was torn. In order to determine accurately the number of pounds of pull required to break the seal and withdraw the wire from the tooth, Rocky Mountain orthodontic wires and tubes were used in the following sizes: 0.025, 0.030, and 0.40 inch.

The tubes were cut to a length at least twice that of the inserted wire. This was done in order to provide ample room to grasp the tube without affecting the sealing agent or its contained wire. One end of the tube was pinched off to permit complete filling of the tube with the sealing agent.

The paste was inserted with an engine-driven instrument (the Lentula). After the tube had been completely filled, the wire to be tested was inserted to its predetermined length. The lengths tested were 10 mm., 15 mm., and 20 mm.

After insertion, the sealer was permitted to set for one week before testing. A Scott tester, as shown, was used for these studies. The bottom of the tube was grasped by the lower clutch and secured. The upper clutch was secured to the protruding wire. By means of an electrically driven motor, the power was developed through a pulley system to create sufficient pull to withdraw the wire from the tube.

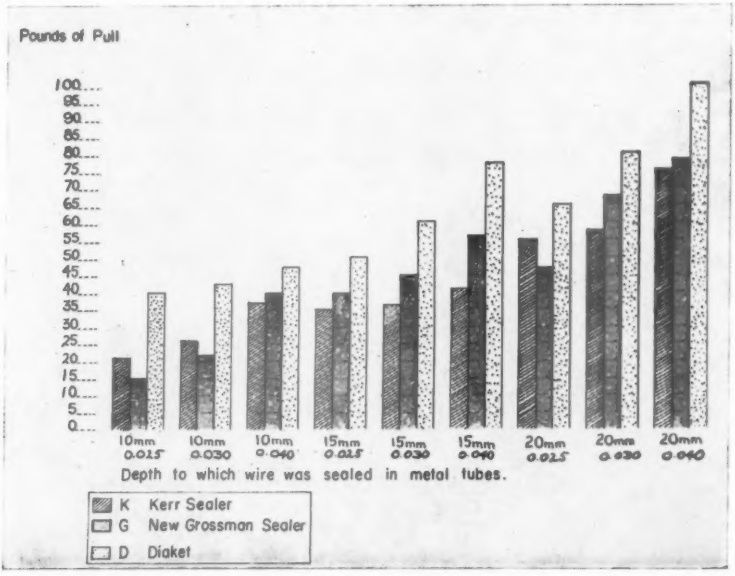
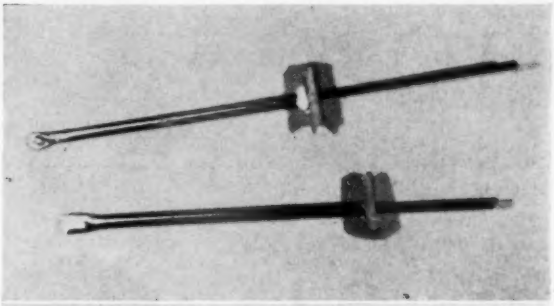


Table III.

A stylus recorded, on a graph sheet, the pounds of pressure required for each measurement. This provided an accurate record of the exact point of breaking the seal.

Results.—Results of these studies are recorded in averages in Tables II and III. It will be noted that as the depth of the wire increased in each tube, the pounds of pressure required increased for each of the sealing agents.

Fig. 11.



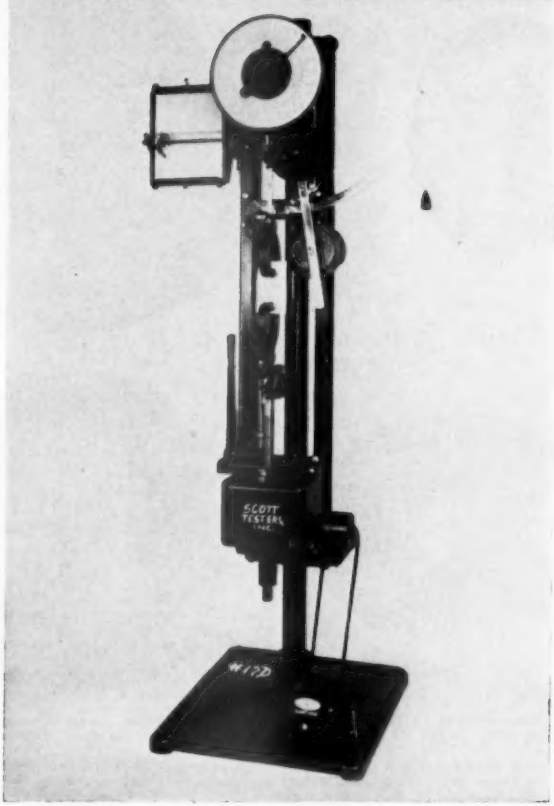


Fig. 12.

Fig. 11.—Orthodontic wires and tubes used in measuring tensile strength of root canal sealers. Note depth markers and pinched tube ends.

Fig. 12.—Scott tester used in tensile strength studies.

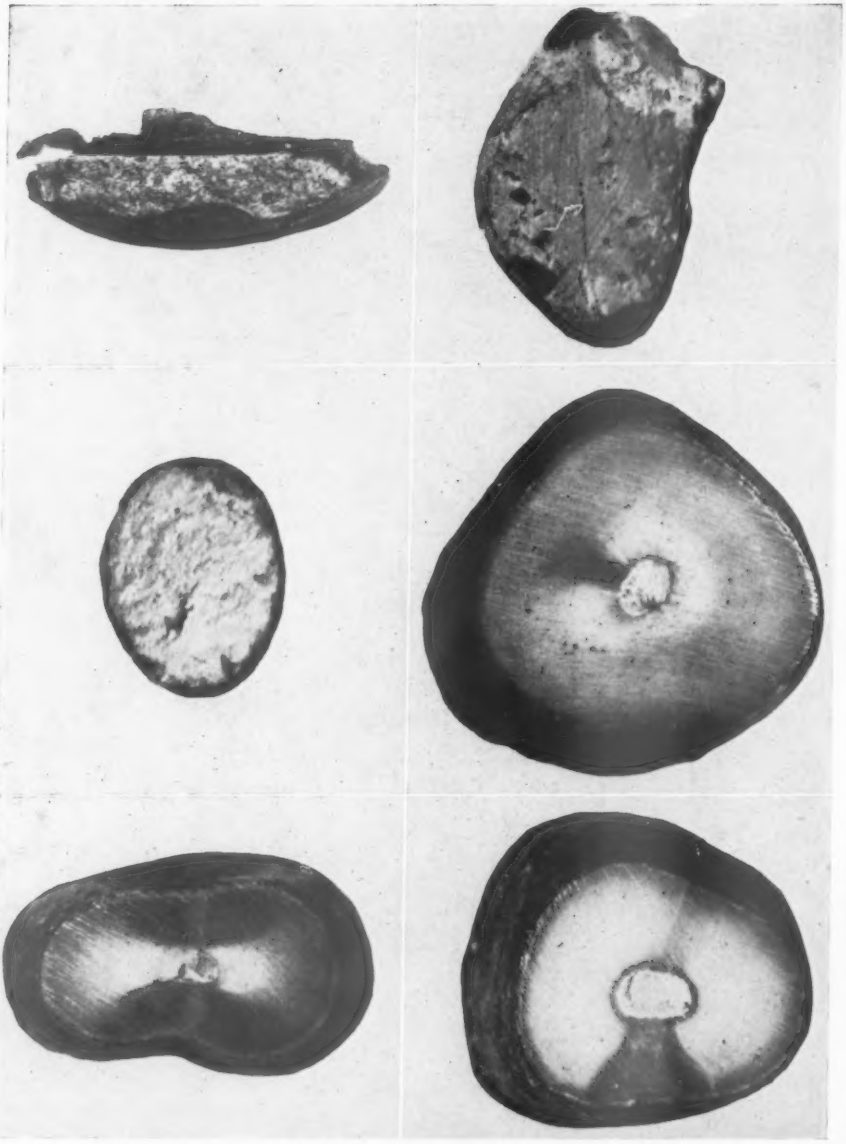


Fig. 13.

Fig. 1

Fig. 15.

Fig.

Fig. 17.

Fig.

Fig. 13.—Cross section of Kerr sealer after immersion in a methylene blue solution for six months. Note outer layer of penetration of dye into the material altering the texture of the sealer.

Fig. 14.—Cross section of the Grossman sealer after six months' immersion in a methylene blue solution. There is very little penetration of the dye substance, and very little alteration may be noted in the texture of the outer surface.

Fig. 15.—Cross section of the Diaket after six months' immersion in a methylene blue solution. There is no penetration of the dye substance, and the outer surface is still hard and glassy.

Fig. 16.—Cross section of tooth filled via lateral condensation of gutta-percha and Kerr sealer, after six months' immersion in a methylene blue solution. Note good adaptation to the canal walls and lack of penetration of dye substance into filling.

Fig. 17.—Cross section of tooth filled via lateral condensation of gutta-percha and the new Grossman sealer, after six months' immersion in a methylene blue solution. There is no penetration of dye substance into filling.

Fig. 18.—Cross section of tooth filled with Diaket and single cone of gutta-percha, after six months' immersion in a methylene blue solution. Note excellent adaptation of sealer to canal walls and lack of penetration of dye into filling.

PERMEABILITY STUDIES

Since an ideal root canal sealer is impermeable to fluids, this property was investigated in the three root canal sealers.

Method.—Several batches of each sealer were prepared and then placed in a methylene blue solution of the type used in staining microorganisms. After intervals of one week, one month, and six months, samples were removed from the solution, dried in the air, and then carefully examined.

In another phase of this study, extracted teeth were used. The root canals were first prepared and then obturated with silver points, gutta-percha points, and lateral condensation techniques, each in conjunction with the three sealing agents being tested.

All of the teeth thus treated were placed in methylene blue baths and permitted to remain in an incubator at 37° C. for a period of six months before they were removed, dried, and sectioned.

Results.—The individual batches of sealing agents, which were removed after one week and one month, were cut in half and showed that the inner surface was free of methylene blue.

In those specimens examined after six months' immersion in the methylene blue, both the Kerr sealer and the new Grossman sealer showed a surface permeability to a depth of approximately 0.5 mm. No permeability was noted in the Diaket material.

To permit observation of the extracted teeth which had been obturated and placed in the methylene blue, cross sections were cut at the apical third and middle of each root and observed under magnification.

It was noted that the methylene blue did penetrate through the dentinal tubules to the root canal, but there was no evidence of permeability through the root canal filling materials studied.

(This article will be concluded in the next issue of the Journal. References for the entire article will appear at that time.)

THE RELATIONSHIP BETWEEN INFECTION AND PATHOLOGY IN THE PULP CANAL AND PERIAPICAL REGION

Lucile S. Smith, Ph.D., Paul R. Thomassen, M.A., D.D.S.,** and Joe G. Sweet II, D.D.S.,*** San Francisco, Calif.*

MANY investigators have reported the presence of various microorganisms in the root canals of teeth with diseases of the dental pulp. However, many of the earlier studies were made on extracted teeth and on teeth of cadavers. Under these conditions, it was not possible to be certain that the organisms obtained had been present in the canals while the teeth were in situ during life. Sommers and Crowley[1] were among the first to use aseptic techniques to obtain specimens from the canals of in situ teeth, but they did not exclude teeth with direct exposures of the pulp canal to the oral cavity. A predominance of anhemolytic and alpha hemolytic streptococci were recovered from cultures collected under these conditions. MacDonald and colleagues,[2] using aseptic techniques and similar methods of entering the pulp chamber but limiting their study to nonvital teeth free from fractures, caries, or restorations, also cultivated bacteria from the pulp. Brown and Rudolph[3] found a high incidence of streptococcus, micrococcus, and diphtheroids and observed microbial forms which were not cultivable in material collected from diseased pulp canals which had no direct connection with the oral cavity.

However, no relationship between the presence of these organisms and the various pathologic conditions which occur in the tooth pulp and periapical region has been established. This report describes the isolation of a number of organisms from diseased pulp canals and the production by these bacteria of various enzymes which have often been associated with pathogenicity and/or invasiveness. Also, an attempt was made to correlate the bacteriologic findings with the roentgenographic interpretations of periapical pathology.

In this study only in situ teeth with pulp involvement and with no direct exposure of the pulp canal to the oral cavity were utilized. The techniques used precluded contamination of the specimens by normal oral flora.

Some of the material included in this paper was presented at the thirty-fifth meeting of the International Association for Dental Research, March 21-24, 1957, at Atlantic City, New Jersey.

These studies were aided by a contract between the Office of Naval Research, Department of the Navy, and the College of Physicians and Surgeons.

This investigation was supported in part by the "Friends of Dr. Charles A. Sweet," Grant for Investigations in Endodontics.

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MATERIALS AND METHODS

Collection of Specimens and Isolation of Bacteria.—In order to facilitate obtaining only organisms present in the pulp tissue, this study was limited to single-rooted anterior teeth with diseased pulps as determined by means of the electric pulp test, thermal tests, percussion, and roentgenographic findings. Those with carious lesions extending into the pulp chamber or with severe periodontal involvement were excluded. In situ teeth were isolated with a rubber dam, using suitable a clamp or clamps, and the entire field was disinfected with a cotton swab saturated with Mercresin. A sterile diamond inverted-cone bur was used to cut a well-defined ledge penetrating deeply into the dentine on the lingual surface of the selected tooth. The area was redisinfected with Mercresin, and a sterile No. 4 round carbide bur was used to penetrate the pulp chamber. The field was disinfected again with Mercresin, care being exercised not to wipe any tooth debris into the cavity.

Two cultures were taken from each tooth in succession, before the use of any chemicals or irrigants, by inserting sterile paper points into the pulp canal as far as possible. One paper point was inoculated into Difco brain-heart infusion broth containing 0.1 per cent agar and 10 per cent ascitic fluid. The second point was inoculated into an enriched cystine broth.* These cultures were incubated at 37° C. until visible growth was observed. The organisms were obtained in pure culture by means of two poured blood agar plates, one incubated aerobically and the other anaerobically (Bray method) at 37° C. Isolated colonies were picked from these plates, and the production of enzymes by the individual strains in pure culture and by combinations of strains isolated from any one tooth was determined. The genus of each organism was determined according to Bergey's *Manual of Determinative Bacteriology.*[4]

Determination of the Production of Enzymes.—

1. *Hemolysins:* Duplicate poured blood agar plates (Difco tryptose blood agar base to which 5 per cent defibrinated sheep blood was added) were incubated—one aerobically and one anaerobically (Bray Method)—at 37° C. After colonies developed, the plates were observed both by the unaided eye and under the low power of the microscope to determine the type of hemolysis produced. The plates were then incubated overnight at 5° C. to allow the development of hot-cold hemolysins, and a second observation was made.

2. *Fibrinolysin:* The production of fibrinolysin was determined by the method of Tillet and Garner.[5] Aliquots of broth cultures containing fibrinolysin prevented the formation of a plasma clot when mixed with oxalated human plasma and calcium chloride.

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*Enriched cystine broth:

Beef extract:	10.0 Gm.	Cystine	60 mg.
Peptone	10.0 Gm.	Distilled water q.c.	1,000 ml.
Yeast extract	2.5 Gm.	pH adjusted to 7.4	
Sodium chloride	5.0 Gm.	Sterilized by autoclaving at 120° C. for twenty minutes.	

3. *Coagulase:* The presence of coagulase in broth cultures was determined by its ability to coagulate rabbit plasma according to the method outlined by Kolmer and associates.[5]

4. *Hyaluronidase:* The method used for determining the presence of hyaluronidase was adapted from that described by Tolksdorf and colleagues.[6] Five-tenths milliliter aliquots of supernates of centrifuged broth cultures were added to tubes containing 0.2 ml. quantities of an aqueous solution containing 0.2 mg. of hyaluronic acid; the contents of each tube were brought to 1 ml. with acetate buffer, pH 6.0, and incubated at 37° C. for forty-five minutes. Control tubes containing 0.2 mg. and 0.1 mg. of hyaluronic acid were similarly diluted and incubated. During incubation the hyaluronidase, if present, hydrolyzed the substrate. After incubation, 10 ml. of 0.9 per cent bovine albumin solution at pH 4.2 was added to each tube to develop turbidities in proportion to the amount of nondepolymerized hyaluronic acid remaining in the tube. The turbidities were read by means of a Coleman spectrophotometer. The hyaluronic acid substrate was extracted from human umbilical cords by the method of Harris and Harris.[7]

5. *Chondroitin sulfatase:* Potassium phenolphthalein disulfate, prepared as described by Whitehead and co-workers,[8] was used as the definitive substrate (Follis[9]). The method of Schultz-Haudt and Scherp[10] was adapted to our use. Enriched broth* to which potassium phenolphthalein disulfate was added to give a final concentration of 0.001M was inoculated with the organism or organisms to be tested and incubated at 37° C. until growth was observed. Then 0.5 ml. of supernatant obtained by centrifugation was added to 2 ml. of 0.4M glycine-sodium-hydroxide buffer at pH 10.4.[11] When sulfatase was present the substrate was hydrolyzed, and a pink color developed.

6. *Proteolytic enzymes (collagenase):*[12] Azocoll hide powder† was prepared according to the method of Oakley, Warrack, and van Heyningen.[13] Two milliliter quantities of sterile filtrates from cultures were obtained by use of the Swinney filter adapted for small Seitz filter pads. These were added to 1 ml. portions of a 1 per cent suspension of azocoll in normal saline, overlayered with sterile mineral oil, and incubated at 37° C. for twenty-four hours. Filtrates from known positive and negative cultures were set up simultaneously as controls. If proteolytic enzymes were present, dye was released from the substrate to develop color in the aqueous solution.

Roentgenographic Interpretation of Periapical Pathology.—

1. *Defined lesions* were those having discernible roentgenographic boundaries.

*Composition of this medium has been shown previously in this article.

†Special fine-grind hide powder was obtained from Frank F. Marshall, Ridgway, Pennsylvania.

2. *Diffuse lesions* were those having irregularly outlined indefinite boundaries which blended into the surrounding bone.

3. *Negative lesions* were those showing no radiolucency at the apex.

4. *Root resorption lesions* were those showing loss of tooth structure at or near the apex.

RESULTS AND DISCUSSION

Ninety cases showing pulp pathology with or without periapical involvement as determined by clinical or roentgenographic diagnosis have been studied. From each of sixty-three cases one, two, or three organisms were obtained. Forty-one strains of corynebacterium, twenty-four of micrococcus, twelve of streptococcus, seven of fusobacterium, two of veillonella, and one each of vibrio, bacteroides, spirillum, erysipelothrix, neisseria, lactobacillus, bacillus, pseudomonas, and yeast were isolated in pure culture.

TABLE I. THE PRODUCTION OF VARIOUS ENZYMES BY STRAINS OF BACTERIA ISOLATED FROM DISEASED PULP CANALS

ENZYME	NO. OF STRAINS TESTED	NO. OF STRAINS POSITIVE
Hemolysins	95	Alpha 15 Beta 36
Fibrinolysin	65	29
Coagulase	86	4
Hyaluronidase	95	25
Chondroitin sulfatase	71	0
Proteolytic enzymes (collagenase)	44	12

As shown in Table I, the following enzymes were produced by some of the organisms: alpha and beta hemolysins, fibrinolysin, coagulase, hyaluronidase, and proteolytic enzymes. However, none of the organisms produced chondroitin sulfatase.

Combinations of organisms from any one case, when grown together, did not produce any enzyme not produced by at least one of the organisms alone.

TABLE II. ENZYMES PRODUCED BY ORGANISMS ISOLATED FROM PULP CANALS OF TEETH SHOWING VARIOUS TYPES OF ROENTGENOGRAPHIC LESIONS

	ENZYMES								
	HEMOLYSINS								
ROENTGENOGRAPHIC LESIONS	ALPHA	BETA	ALPHA AND BETA	ANHEMOLYTIC	FIBRINOLYSIN	COAGULASE	HYALURONIDASE	CHONDROITIN SUFATASE	PROTEOLYTIC ENZYMES (COLLAGENASE)
Defined	7/36*	14/36	1/36	14/36	12/20	1/32	11/36	0/25	8/16
Diffuse	4/18	10/18	0/18	4/18	8/15	2/15	12/18	0/14	2/9
Negative	1/9	2/9	2/9	4/9	5/7	1/9	1/9	0/7	2/4
Root resorption	2/12	5/12	0/12	5/12	6/7	1/10	5/12	0/8	2/6

*Number of cases positive over number of cases studied.

The production of these enzymes by the organisms indicates that many of the organisms present in root canals are potential pathogens and are capable of producing lesions.

Attempts to correlate the type of periapical radiolucency with the exoenzymes produced by organisms isolated from the involved teeth showed that no one enzyme or combination of enzymes was uniformly associated with a specific type of roentgenographic lesion. Neither was there any correlation between the enzyme or enzymes produced and the presence of root resorption (Table II).

From twenty-seven of the ninety cases studied, no cultivable organisms were obtained. Only one of these showed a diffuse periapical lesion, fifteen were from the defined group, and eleven were from the negative group. The fact that such a large proportion of the cases were in the defined and negative groups suggests that the type of roentgenographic picture may reveal different stages in the development and control of the lesions. It is possible that future studies will show that the defined roentgenographic lesion represents a controlled infectious process comparable to the circumscribed or localized lesion found in other parts of the body.

SUMMARY

Root canal material obtained asceptically from ninety teeth with diseases of the dental pulp was cultured. The pulp chambers of these teeth had no direct exposure to the oral cavity.

Ninety-five strains of organisms were obtained in pure culture. Fifteen produced alpha hemolysin, thirty-six produced beta hemolysin, twenty-nine produced fibrinolysin, four produced coagulase, twenty-four produced hyaluronidase, and twelve produced proteolytic enzymes.

No one enzyme or group of enzymes was associated with any one type of roentgenographic lesion.

The authors wish to express their appreciation to Drs. John K. Young and Bertram Wolfsohn for valuable counsel and to acknowledge the technical assistance of dental students George Louie, Allen D. Miller, and Robert L. Ratcliffe.

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BACTERIOLOGY

AIR HYGIENE IN DENTAL PRACTICE

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INTRODUCTION

THE problem of disease transmission through the hands, discussed previously,[21] is relatively simple and manageable in comparison to the problem of transmission of respiratory diseases through the air. The fact that inhalation into the respiratory tract is the most common means by which infectious agents enter the body makes this an especially serious problem.

The possibility of air-borne infection had been recognized in the early days of aseptic surgery. Lister's development of carbolic acid spraying in the operating room is a familiar story. This aspect of asepsis became neglected, however, as attention shifted to other problems of microbial control. About twenty-five years ago, when it became clear that uncontrolled surgical infections were still occurring at an embarrassingly high rate, attention returned to the problems of air hygiene. The important advances which have since been made[34] are of considerable interest to the dentist.

MECHANISMS OF MICROBIAL SPREAD THROUGH THE AIR

The spread of air-borne microorganisms occurs either (1) *directly* by the movement of comparatively large droplets from the mouth or nose of one person to another person or (2) *indirectly* by droplet nuclei formed from small droplets which evaporate and remain floating in the air. Strictly speaking, the first mode of spread is a form of contact transmission, whereas the second is true air-borne transmission. The eventual fate of the droplets or droplet nuclei, if they fail to reach the respiratory tract, is sedimentation upon surfaces; however, they may later become resuspended by drafts, movement of persons, dusting, or other activity.[11]

During ordinary expiration under normal conditions, the expired air will yield only one to two colonies in twenty breaths when a blood agar plate is

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held in front of the mouth.[1] Loud laughing expels only a few organisms, but the loud speaking of such sharp consonants as *P, B, T, F,* and *K* will throw out a large number of bacteria through the teeth. Speaking softly reduces the number of droplets by almost 75 per cent.[12]

An average vigorous sneeze[7] causes the emission of about 100,000 particles of a size small enough to remain in the air for more than one minute. Of these, about 4 per cent usually will remain suspended in still air for as long as thirty minutes. Two secretions are involved in a sneeze[15]: (1) fluid nasal secretion, which is not propelled violently into the air, and (2) oral secretion, which is expelled at high velocity and forms the chief component of expelled material. Large droplets expelled during sneezing may easily be thrown five to nine feet,[15] while droplet nuclei may travel much farther and may persist in the air for quite long periods of time. The present evidence[33] indicates, however, that direct contact via large droplets plays a greater role in the spread of upper respiratory disease than droplet nuclei.

Ordinarily, coughing does not discharge saliva. Since the cough starts in the back of the throat, it disperses secretions of the pharynx, secretions of the nose if they have dripped back into the throat, or material from below the larynx.[15]

It appears that the site of droplet production, and particularly of the small droplets which remain air-borne as droplet nuclei, is almost entirely in the front of the mouth.[12] The primary site of colonization for beta hemolytic streptococci is the throat and for pathogenic staphylococci it is the nose, but it is known[20] that in many cases these colonizations may occur simultaneously in the mouth as well. A comparison[35] of the cultivable bacteria in the mouth, pharynx, and nose has shown that the flora of the mouth and pharynx is more closely related than that of the mouth and nose or of the nose and pharynx. There are no data on the occurrence or concentration of viruses in the saliva as compared to those in the nose or pharynx.

THE RISKS OF AIR-BORNE INFECTIONS IN DENTAL PRACTICE

Since the dentist works so close to his patient, it is apparent that such simple and common activities as talking, coughing, and sneezing involve a greater risk of direct exchange of oral and pharyngeal microorganisms than is the case in ordinary interpersonal relationships. In addition, there are certain dental procedures which may be the source for infective droplets and droplet nuclei.

When the front teeth of a patient are subjected to short blasts of air for one minute,[1] blood agar plates held directly in front of the mouth will show an average of about 170 colonies. Of these, about 70 per cent are alpha hemolytic streptococci, and the remainder consist of *Staphylococcus albus* and nonpathogenic spore formers. The air blast itself, unless a filter is used, will contain bacteria from the room air.

If burr shavings are blown out from a cavity preparation with a blast of air,[2] blood agar plates held directly in front of the mouth will show counts of between 6 and 400 colonies per plate. The organisms found here are, again,

largely alpha hemolytic streptococci, with nonhemolytic staphylococci, yeasts, lactobacilli, and neisseria also present in large numbers. It is possible that burr shavings may cause pathologic changes in the lungs upon inhalation, but no evidence is on hand to suggest that this has ever occurred. Nevertheless, however remote the possibility may appear, it is a point to keep in mind.

When water spray from the dental unit is applied for twenty seconds against the mouth,[1] blood agar plates exposed in front of the mouth show an average of 500 colonies per plate. These are mostly alpha hemolytic streptococci and staphylococci. Small numbers of sarcinae, yeasts, aerobic spore formers, neiseria, and coliforms are also found. From culture plates, therefore, it appears that water spray may result in a considerable number of microorganisms being thrown out of the mouth into the air of the room.

Studies of water spray particles caught on glass slides give comparable results. It has been shown[5] that a glass slide held ten inches before and slightly above the mouth for less than five seconds while Vincent's infection lesions are washed by a water spray will show a great many large spots consisting of a mat of Vincent's organisms. The spray may be readily felt by the patient on his face and by the dentist on his arms. It appears highly probable, from these observations, that the breathing space of the dentist is invaded by the aerosol created by the use of the water spray in the mouth.

The findings of the bacteriologic study of drinking fountains by Mallmann and Fontes[27] support the suspicion that the bowl with running water used for disposing of the patient's saliva at the dental chair may be an additional source of aerosol production. These investigators found that the normal and correct use of drinking fountains created aerosols which rapidly contaminated all parts of the fountain, including the orifice and the air above the fountain. The aerosols were formed as a result of the splashing of the contaminated water rinsed from the lips and mouth of the user.

The front of the clothing of the operator himself becomes contaminated[18] from the mouth, nose, and throat flora of the wearer as well as from the patient. This later may become the source of contaminated dust.

THE CONTROL OF AIR-BORNE INFECTIONS IN DENTAL PRACTICE

Face Mask.—The face mask has been recommended occasionally for the mutual protection of the patient and the dentist. (See reference 10 for a good summary of the place of this technique in dentistry.) The conventional type of surgical mask acts as both a filter and a deflector, but recent studies[9, 14] have shown that it is surprisingly inefficient. Blood agar plates held eighteen inches from the face without any mask, with one mask, and with two masks gave the following plate counts during breathing and talking:[9]

SUBJECT	NO MASK	ONE MASK	TWO MASKS
A	15	4	3
B	11	12	8
C	7	3	5

It would appear[10] that the purely deflector mask may be the most suitable in dental practice. This type consists of a solid sheet of clear transparent plastic, held over the operator's face, which deflects mouth spray from both the operator and the patient. It allows a free flow of air without filtration and is comparatively comfortable to use. A number of designs have been developed and are nicely discussed by Docking and Amies.[10]

When operative procedures permit, the use of a rubber dam is an effective means for preventing the spraying of the patient's saliva. The dentist can control his own production of salivary aerosol by minimal and soft speaking in addition to the use of a face mask.

Air Conditioning.—It is possible to control, to some extent, the bacterial as well as the allergenic contamination of air in a room by a number of methods which, collectively, may be regarded as a part of air conditioning: (1) mechanical ventilation, (2) filtration, (3) disinfection by ultraviolet irradiation or chemical aerosols, and (4) dust suppression. Complete air conditioning also includes (1) heating, (2) cooling, (3) drying, and (4) humidifying.

Ventilation and Filtration.—Rooms in which light work is performed should have not less than 900 cubic feet of outside air per person per hour[37] or two air changes per hour, whichever is greater. This is of the first importance in reducing air-borne infection. The quality of the air can be improved considerably by filtration, which is an aid in the removal of both allergens and nonpathogenic microorganisms in the outside air.[16] An efficient system of filters can remove as much as 99.5 per cent of the bacteria in the air.[8]

The filtration of pollen from the air is not as efficient with the mechanical filters as with precipitator filters.[29] In the case of mechanical filtration, the relief obtained by a hay fever sufferer depends entirely upon his degree of sensitivity, since all the pollen is not removed. There are some mechanical filters which will cut ragweed pollen counts down to 7 per cubic yard of air when the outside count is 200 per cubic yard. Spores of hormodendrum and alternaria are not removed nearly as effectively.[31]

Although the organisms given off in the room by its occupants may be efficiently removed by a good ventilating system, the air may simply be drawn into another room to give increased contamination if the route of air circulation is not carefully checked out. When the air is simply recirculated by a heating or air conditioning system, we then have an efficient mechanism for the uniform distribution of all the bacteria expelled into the air.[26]

Ultraviolet Irradiation.—Additional control of the microbes in the air may be obtained with ultraviolet irradiation,[17] which is especially effective against the microorganisms ejected from the mouth and upper respiratory tract.[24, 36] Field studies[22] have shown that under barracks conditions a reduction of approximately 20 per cent in the incidence of febrile respiratory admissions may be obtained from the use of ultraviolet irradiation. Unexplained infections in clean wounds following surgery have been reduced to from 5 to even

1 per cent of previous levels.[19] Apparently, similar studies have not been made in the dental operating room, but, in view of the possibility of droplet and droplet nuclei transmission, such studies at least merit attention and evaluation.

The requirements for the successful application of ultraviolet light to the disinfection of air are (1) supplementing irradiation with dust suppression measures, since ultraviolet light is not efficient against bacteria and viruses protected by dust and (2) attaining a radiation of sufficient intensity but not so high as to affect the eyes or skin of occupants.[30] Normal ceiling paints and plasters reflect very little ultraviolet light; therefore, if the lamps are not in direct vision, no goggles or special protective clothing need be worn.

With a correct ultraviolet installation, the air in the upper part of the room is irradiated and maintained in a high degree of sterilization.[4] The air below the seven-foot level is kept at a low bacterial count by being continually diluted with purified air from the upper strata.

The cost of ultraviolet irradiation is nominal. A single 30-watt germicidal lamp in a suitable fixture for upper-air irradiation is adequate for a floor area of about 200 to 300 square feet, if properly designed and installed. The cost of operation, including lamp replacement and amortization of the original cost of the lamp, was about $1.00 per month at 1949 prices.[25]

Disinfectant Vapors.—There have been developed chemical aerosols of germicidal agents, including propylene and triethylene glycols, which also give significant reductions in the bacterial content of air.[34]

Among the limitations of both radiant and chemical air sterilization is the fact that neither procedure has a demonstrable effect on infectious agents deposited upon environmental surfaces or fabrics, which may form "secondary reservoirs" for the redispersal of pathogens into the air.

Dust Suppression.—A number of techniques and precautions have been developed in an effort to control the redispersal of microorganisms after emission and sedimentation. Walls should be designed to prevent dust accumulation. They should be smooth, with rounded corners; there should be no horizontal ledges, even at doors; furniture should be built in, with as few movable pieces as possible. Dry dusting and dry sweeping of floors should not be done. The most effective method of keeping down dust from floors is to use oil on wood floors or on linoleum. Oiling is unsuitable for terrazo, cement, or asphalt, but wet sweeping or sweeping with sawdust moistened with calcium chloride is helpful. Vacuum cleaning is better than dry dusting, although large numbers of microorganisms pass through the walls of the dust bag of the ordinary type of upright vacuum cleaner. Floor washing from a bucket is most unsatisfactory.[3] Daily application of germicide to a floor surface gives a significant reduction in the number of microbes on the floor surface.

Quite recently, germicidal floor waxes have been placed on the market.[32] Preliminary studies indicate that these materials are able to eliminate small numbers of organisms from wax-coated surfaces in simulated sneezing experiments.

Humidity and Temperature.—High relative humidity in combination with high temperature[28] has a significant effect in precipitating and maintaining perennial respiratory allergy.

It is recommended[37] that an air temperature of between 68 and 72° F., with moderate or natural humidities, be maintained. In warm weather, temperatures between 75 and 80 degrees, with relative humidity of less than 50 per cent, are best.

Relative humidity alone in narrow intermediate ranges around 50 per cent is lethal to air-borne microorganisms. Humidities above or below these ranges have no such germicidal effect. Among the organisms susceptible in this way are the pneumococcus, staphylococcus, streptococcus, and the influenza virus.[23] The lethal effect of humidity appears to be dependent upon the presence of sodium chloride in the saliva.

CONCLUSION

A survey of the problem of air hygiene with special reference to dental practice reveals that the dentist carries on a number of activities and procedures which involve real risks of aerial transmission of disease. The methods available for the practice of air hygiene in the dental office are discussed.

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ORAL ANATOMY AND HISTOLOGY

HISTOGENESIS OF THE KERATINIZED ENAMEL CUTICLE

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INTRODUCTION

THE thin, transient, keratinized enamel cuticle, also referred to as Nasmyth's membrane, overlying the crown of the developing tooth has been the object of numerous investigations since the days of Huxley (1853).[7] There has been considerable divergence of opinion as to its origin, histogenesis, structure, and function. An excellent review of the literature was presented by Chase,[3] who contended, from his own observations, that all component parts of the enamel organ, except the enamel pulp, contributed to the histogenesis of Nasmyth's membrane. Chase, moreover, held that Nasmyth's membrane consisted of an inner pellicle formed from the fused cuticular borders of the ameloblasts and an outer cellular portion composed of remnants of the enamel organ and the gingival epithelium. Contrariwise, his complete review of the literature up to 1926 shows that other investigators have believed this structure derived, individually or collectively, from the outer enamel epithelium, ameloblasts, membrana performativa, or connective tissues since it was supposed by some to be composed of cementum. Further complications were introduced by Gottlieb,[5] when he suggested that there were two cuticles instead of one. Gottlieb held that prior to the stage of eruption the ameloblasts elaborate a thin "primary cuticle" about 1 micron in thickness which later may calcify. At the time of eruption, however, a "secondary cuticle" is elaborated from the reduced enamel epithelium. The reduced enamel epithelium which produces this cuticle, he holds, is composed of the stratum intermedium and ameloblasts. Along the surface of the root, he contends, the reduced enamel epithelium becomes the epithelial attachment. The work of Waerhaug,[12] who was able, it seems, to induce a membrane corresponding to the "primary cuticle" to appear on the surface of acrylic crowns and cellulose acetate films, throws grave

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This investigation was aided in part by Grants #C-2621 Pathology and #5182, United States Public Health Service.

doubt on the existence of the "primary cuticle." In 1956 we[8] demonstrated in swine embryos that the epithelial attachment is a derivative of the stratum intermedium and does not arise from the enamel epithelium, as defined by Gottlieb, in human material. With such diversity of opinion still extant concerning the origin and structure of the enamel cuticle, we undertook an analysis of the problem as found in developing swine embryos. We realize that diverse animals are involved in the above-mentioned controversy; yet, pig dentition is so closely related to the human dentition that the results obtained indicate events that probably also occur in the human being. We feel that the relative phylogenetic proximity of swine to the human family affords an opportunity to explore processes in development that may apply to the human situation. The related processes of tooth development in the human being, coupled with the environmental factors of mouthing and sucking prior to and during tooth eruption, complicate an adequate interpretation of events in development. Tissues in the human jaw just prior to eruption of the teeth may become greatly disorganized. With this in mind, it was felt that a study of events involved in the elaboration of the enamel cuticle in an animal which has undisturbed tissues prior to birth might throw valuable light on this controversial subject.

MATERIAL AND METHODS

Advanced stages in embryonic development of the deciduous teeth in fetal pigs was selected for this study. Stages at intervals from 200 mm. to 260 mm. C.R. measurement were removed from the uterus. With a sharp heavy knife, anterior portions of both the upper and the lower jaws were removed and bisected on either side of the canine teeth and then plunged into diverse fixatives, including Bouin's, Carnoy's, and 5 per cent trichloroacetic acid in 80 per cent alcohol. After proper washing, several procedures were adopted. Some blocks of tissue were immediately decalcified in Versene, the tetrasodium salt adjusted to pH 7.4; others were decalcified in 3 per cent nitric acid, while in others the enamel was literally shelled away from the investing membranes. The embedding procedure generally adopted, however, consisted of celloidin embedding by the slow method, followed by decalcification of the embedded block in 3 per cent nitric acid. This permitted examination of the tissues intact without disturbing the cuticle in its relationship to the organic remnants of the enamel and other soft tissues. Sections were cut on a sliding microtome and individually handled in the staining process. Sections were stained with Papanicolaou stain.[9] With this stain, keratin appears yellow, connective fibers and ground substance appear green, and nuclear and cytoplasmic elements stain red to blue. The sulfhydryl reactions of the ferricferricyanide method were used to determine SH groups. Sodium thioglycolate treatment was utilized for conversion of SS to SH groups.[10] The Barrnett-Seligman[10] reaction was also used but with less success, since the celloidin sections were softened and greatly distorted. Despite the careful treatment afforded by the celloidin method, it was with the greatest difficulty that the fragile enamel cuticle could be preserved, especially at the extreme coronal aspect.

OBSERVATIONS

An examination of the lower jaw of a 240 mm. CR. pig embryo shows that the teeth are already in an advanced stage of eruption. The superior aspect of the lower jaw (Fig. 1) shows three teeth in active stages of eruption, the most advanced being the canine, which in the pig gains ascendancy and ultimately develops into the prominent tusk. The relationships of the oral mucosa to the gingivae and protruding enamel crowns are clearly set forth and present an ideal situation for a study of the intimate relationships of the tissues involved in the histogenesis of Nasmyth's membrane. A labiolingual decalcified celloidin section in situ is illustrated in Fig. 2. A well-developed root with cementoblast and cementocyte activity is evident. Since this section is from a celloidin-embedded block with subsequent decalcification, all relationships are undisturbed except for the loss of calcified enamel at the cervical region of the crown. That this is a true state of affairs may be ascertained from an examination of the coronal sections of the two teeth illustrated in Figs. 5 and 7. Fig. 5 presents a ground section of the cusp of a tooth which shows the relatively small amount of enamel in relation to the extensive dentine. Fig. 7 shows a comparable section decalcified after celloidin embedding. There is no evidence of shrinkage or loss of relationships. The fact that the decalcification of the enamel was more evident at the cervical region of the tooth can be interpreted in no other way than to hold that this region is more highly calcified than the more occlusal areas. Ameloblasts remain in a high state of activity along the cervical region of the crown opposite the enamel space, continuing to lay down enamel long after the eruption of the tooth. At this locus the ameloblasts are nearly parallel with the rods, in contrast to their greater inclination on the more occlusal aspects. At this stage there is no evidence of a well-defined periodontal membrane. A section taken more coronally at the upper limits of the enamel space, as illustrated in Fig. 3, reveals (from left to right in the photomicrograph) flattened superficial cells of the stratified oral epithelium overlying deeper polyhedral cells with marked intercellular bridges. Subjacent to the oral mucosa is a narrow band of connective tissue with a large adjacent elongated blood vessel indicative of a good blood supply. Next in order is the five- or six-layer stratum intermedium, the cells of which are now elongated incisally along the base of the acutely inclined ameloblasts. It would appear that as the tooth continues to erupt there is a constant pull on the cells coronally, thus elongating all the cellular elements involved. Remnants of the organic constituents of the enamel are still evident. The cells making up this whole complex have a marked affinity for the hematoxylin of Papanicolaou's stain with no registration at all for the orange G component, indicating that no keratin has formed at this time.

In a region located more coronally, as illustrated in Fig. 4, it is observed that the connective tissue component has disappeared from the complex and that the oral mucosa has formed a union with the stratum intermedium and ameloblasts. Here we have a complete picture of the united epithelium. Fig. 6, a section taken still more occlusally, shows that the single layer of cuboidal

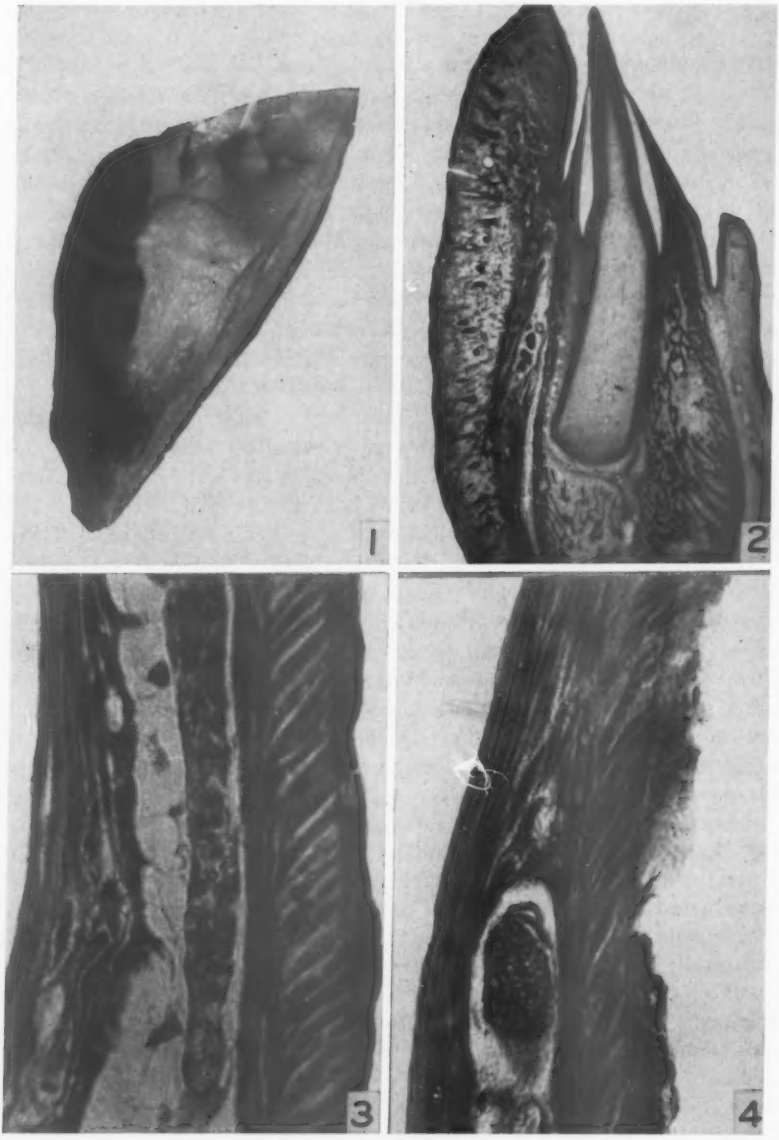

Fig. 1.—Portion of mandible of pig embryo, 260 mm. C.R. measurement showing erupted teeth, with gingivae and crown. (Magnification, ×3; reduced 1/10.)

Fig. 2.—Labiolingual section of tooth in alveolus. Note investing membranes over crown of tooth. Celloidin-embedded and subsequently decalcified. (Magnification, ×9; reduced 1/10.)

Fig. 3.—High-power view of section of crown midway between cusp and cervical region. Note oral mucosa to left, connective tissue, stratum intermedium, and ameloblasts. (Magnification, ×880; reduced 1/10.)

Fig. 4.—High-power view of section closer to cusp showing more intimate fusion of layers. (Magnification, ×880; reduced 1/10.)

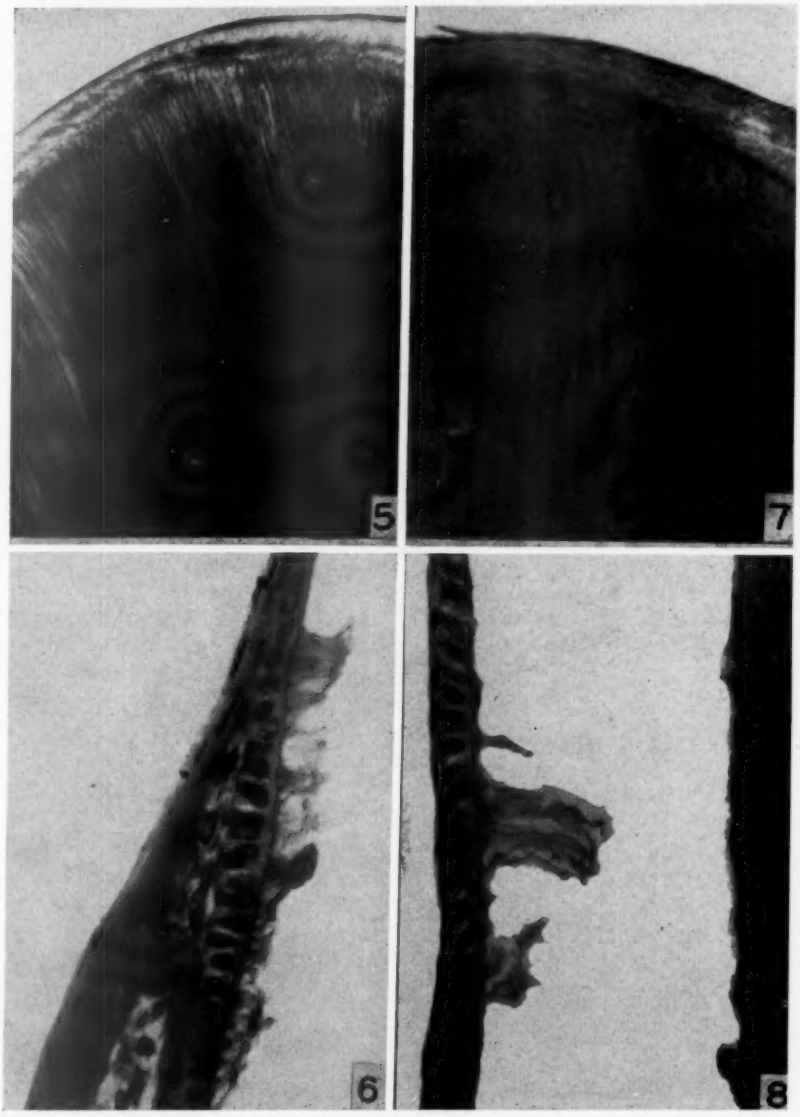

Fig. 5.—Ground section of cusp showing thin enamel and thick dentine. (Magnification, ×800; reduced 1/10.)

Fig. 6.—High-power view of section of investing layers near cusp showing few cells of oral mucosa and few cuboidal ameloblasts. (Magnification, ×450; reduced 1/10.)

Fig. 7.—Section similar to Fig. 5, except decalcified. Note lack of shrinkage. Celloidin-embedded. (Magnification, ×800; reduced 1/10.)

Fig. 8.—High-power view of section of celloidin-embedded tooth. Note single layer of oral mucosa overlying cuboidal ameloblasts with adherent organic enamel matrix. (Magnification, ×450; reduced 1/10.)

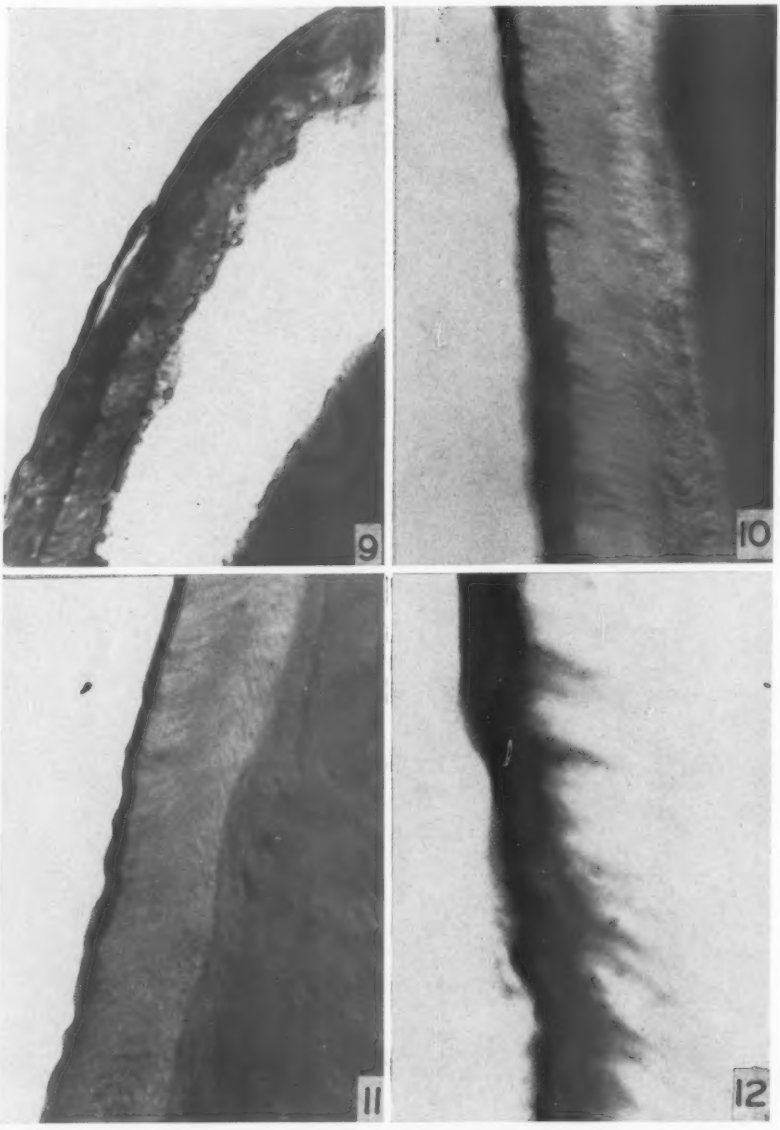

Fig. 9.—High-power view of section near cusp showing dentine, enamel space, enamel matrix, ameloblasts with pyknotic nuclei, and single layer of sloughing squamous cells. (Magnification, ×800; reduced 1/10.)

Fig. 10.—High-power view of section of cuticle and interrod substance possessing marked affinity for orange G. (Magnification, ×1,200; reduced 1/10.)

Fig. 11.—High-power view of section of enamel cuticle, enamel rods, and dentine. (Magnification, ×800; reduced 1/10.)

Fig. 12.—Oil-immersion photomicrograph of enamel cuticle giving SH reaction. (Magnification, ×1,500; reduced 1/10.)

ameloblasts lies in juxtaposition to the flattened cells of the oral mucosa, while the stratum intermedium has become reduced to a layer two or three cells in thickness. Proceeding still more coronally (Fig. 8), all that can be observed is a single layer of ameloblasts overlaid by a single layer of oral mucosa. There is no indication of an enamel cuticle, either primary or secondary, between the ameloblasts and the enamel at this locus, on the one hand, or between the enamel and the oral mucosa on the other. An examination of this photograph shows that the single layer of flattened squamous cells could not elaborate the thick cuticle indicated in Figs. 10 or 11. However, the ameloblasts at this stage, although reduced to a cuboidal state, appear to have a well-formed nucleus and are still active. They further appear to be the only possible source from which the cuticle can be produced. An analysis of sections in a still more coronal position, as illustrated in Fig. 9, shows that the ameloblasts have become highly pyknotic with loss of clear cellular boundaries. The single superficial squamous-cell layer is rapidly becoming detached from the subjacent ameloblasts.

The final stage to which we direct our attention is that illustrated in Fig. 11, in which a well-formed orange G-positive cuticle intimately attached to the subjacent enamel rods is finally elaborated. Extensions between the enamel rods (Fig. 10) likewise have a marked affinity for the orange G. We thus have a well-formed cuticle established over the coronal aspect of the tooth which subsequently will be further elaborated along the cervical region of the enamel. This structure originates through an actual conversion of the ameloblasts into a continuous, deeply staining orange G-positive cuticle rather than as a secretion product of the ameloblasts. While components of the enamel likewise have a marked affinity for the orange G stain, as illustrated in Fig. 10, these observations do not necessarily indicate that the extensions into the enamel matrix are derived from the cuticle itself; rather, it would appear that the organic components of the enamel which stain with orange G, while still the product of ameloblastic activity, are elaborated at an earlier time and keratinize simultaneously with the cuticle.

Sulfhydryl reactions indicative of the presence of keratin in the cuticle and interrod substance give a pronounced SH reaction (Fig. 12), while portions here and there remain negative but likewise give a pronounced SH reaction with sodium thioglycolate treatment. That the cuticle is made up of keratin seems self-evident.

DISCUSSION

In this study we have extended our previous observations on the role of the stratum intermedium in tooth development[8] with reference to the detailed aspects of the developing enamel cuticle. While we realize that there may be divergencies between those events occurring in the pig and in the human being, we believe that the processes involved here are comparable and that applications may be made to those occurring in the human subject. It would appear that the structure in question should not be referred to as a membrane in the strictest sense of

the word, since its keratinous nature would preclude that nomenclature. Rather, we concur with Andrews[1] that the structure should be referred to as the enamel cuticle. It would be more valuable, we suggest, to name this structure "Nasmyth's cuticle" rather than "Nasmyth's membrane," as "Nasmyth's cuticle" would connote more accurately what the structure really is.

We find no evidence of a differentiation of two cuticles as maintained by Gottlieb, nor do we find any evidence for the origin of the single enamel cuticle from any layer or combination of layers other than the ameloblasts themselves. The elaboration of the interrod substance is not related per se to the elaboration of the cuticle, although both are undoubtedly keratinized structures. While both the cuticle and the interrod substance are products of ameloblastic activity, the interrod substance is laid down prior to the elaboration of the cuticle itself and they both appear to be keratinized simultaneously. Both the cuticle and the interrod substance become highly SH positive, whereas the more mature areas of the cuticle appear to possess SS groups. The cuticle is of a keratogenous nature and is not cementum, as earlier supposed. It is readily identified by its marked affinity for the orange G stain and marked SH-SS relationships. We believe that the evidence presented by Gottlieb for the presence of a "primary" calcifying cuticle, as postulated through a microscopic analysis of a structure as small as 1 micron in thickness, is highly tenuous and that this structure may be merely an organic component of the ameloblasts themselves. It may be nothing other than that shown by us[2] in Figs. 10 and 11. We recommend a return to the more simple explanation of the enamel cuticle.

CONCLUSIONS

1. There is but one cuticle covering the enamel, demonstrable in the pig's deciduous canine tooth.

2. This cuticle is produced by conversion of the degenerating ameloblasts, and no other constituent layer enters into its formation.

3. It is keratogenous in nature.

4. Its function in tooth development is, practically speaking, of little consequence and plays little or no part in the role of a protective membrane; it is, however, the natural consequence of epithelial cells to keratinize.

5. It seems more reasonable to refer to this structure as an enamel cuticle than as a membrane. We suggest that it be called "Nasmyth's cuticle."

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*The formula for Papanicolaou's EA36 is incorrect as it appears in this paper. The authors in no way modified the original formula given in Lillie's[10] book.

PROFESSIONAL NEWS ITEMS

Association of American Women Dentists

The Association's annual meeting will be held in Dallas, Texas, on Monday, Nov. 10, 1958. Headquarters at the Baker Hotel and will be open on Sunday, November 9, for visiting women dentists.

Fifth National Dental Congress of Cuba

The fifth National Dental Congress of Cuba will take place March 29 to 31, 1959, instead of Nov. 14 to 17, 1958. Headquarters will be the Casa de la Cultura, located at Avenida de los Presidentes and Calle Tercera, Vedado, Havana.

For further information, please contact the secretary, Dr. Jesus Lastra Martinez, Colegio Estomatologico Nacional, Calle L No. 353, Vedado, Havana, Cuba.

Department of Stomatology School of Medicine, Boston University

The Oral Surgery Section of the Department of Stomatology, Division of Graduate Studies, School of Medicine, Boston University, wishes to announce a course in oral surgery for one academic year beginning Oct. 1, 1958. It is designed to present a basic review of the clinical phases of oral surgery coordinated and correlated with studies in the fundamental sciences as they apply to this field. The course is planned to provide a foundation for additional clinical training leading to qualification for the practice of oral surgery as a specialty. It will be under the direction of Drs. Kurt H. Thoma and Edward Sleeper.

A complete course schedule is available on request from the Department of Stomatology, Division of Graduate Studies, School of Medicine, Office of the Dean, Boston University, 80 East Concord St., Boston 18, Massachusetts.

The Periodontia Section of the Department of Stomatology, Division of Graduate Studies, School of Medicine, Boston University, wishes to announce a two-week course in periodontal therapy to be held Oct. 6 to 17, 1958. The purpose of the course is to acquaint the practitioner with the examination, diagnosis, and etiology of periodontal diseases, so that he may have an understanding of the various objectives of therapy. Each operative procedure will be discussed as to methodology, indications, and contraindications; case planning will be emphasized. The course includes lectures, demonstrations, and seminars. It is under the direction of Drs. Henry M. Goldman and Bernard S. Chaikin. Enrollment fee is $350.00.

Those interested may write to the Department of Stomatology, Division of Graduate Studies, School of Medicine, Office of the Dean, Boston University, 80 East Concord St., Boston 18, Massachusetts.

New York University College of Dentistry

An eight-month postgraduate course in pedodontics will be offered again during the 1958-59 academic year. The course, which will be presented from 1 to 4 P.M. on Wednesdays from September 17 through May 20, covers all aspects of pedodontics. Instructing will be Dr. Harold K. Addelston, Professor and Chairman of the Department of Pedodontics, and Dr. Sidney M. Kronfeld, Associate Professor of Pedodontics.

Further information may be obtained from Director, Postgraduate Division, College of Dentistry, 209 East 23rd St., New York 10, New York.

University of Pennsylvania

The following postgraduate courses will be offered:

Endodontics. Eleven one-day sessions Wednesdays, beginning Oct. 1, 1958. Dr. Louis I. Grossman and associates.

Histopathology of the Dental Pulp. Oct. 13 to 17, 1958. Drs. Samuel Seltzer, I. B. Bender, and associates.

The Use of High-Speed Instruments and Equipment. Oct. 15 and 16, 1958. Drs. Amsterdan, DeRevere, Gabel, and Kilpatrick.

Complete Denture Prosthesis. Oct. 27 to 31, 1958. Dr. H. Milton Rode.

For further information, please write to Postgraduate Courses, School of Dentistry, University of Pennsylvania, 4001 Spruce St., Philadelphia 4, Pennsylvania.

Temple University School of Dentistry

A course in clinical endodontia will be presented Oct. 20 to 25, 1958, by Drs. Leonard Parris and Herbert Schilder and their associates. This is an intensive participation course designed to prepare the dentist to meet common endodontic problems. Tuition is $175.00. The class will be limited to ten.

For additional information, write Dr. Louis Herman, Temple University, School of Dentistry, 3223 North Broad St., Philadelphia 40, Pennsylvania.

Western Reserve University

The School of Dentistry announces the following courses:

Appearance and Function in Full and Partial Prosthesis. Sept. 13 and 14, 1958. Fee, $75.00. Dr. Earl Pound.

Minor Oral Procedures and Emergency Treatment in the Dental Office. October, 1958. Fee, $50.00. Drs. Ackell, Spilka, and Tomaro.

Endodontics. Nov. 6 to 8, 1958. Fee, $75.00. Dr. Louis I. Grossman.

Periodontal Prosthesis. Jan. 3 to 5, 1959. Fee, $75.00. Drs. Morton Amsterdam and Walter Cohen.

Psychodynamics and Practice Administration. March 30 to April 1, 1959. Fee, $75.00. Dr. Lloyd Hollander and staff.

Philip Burwasser, D.D.S., Ph.D.
Director of Postgraduate Education
Western Reserve University
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First Argentine Congress of Stomatology

The Federación Odontológica de la Provincia de Buenos Aires is organizing the first Argentine Congress of Stomatology, which will be held Oct. 1 to 4, 1958, in Mar del Plata. Colleagues in the United States are invited to participate. The principal subjects will be "The Problem of Cancer in Odontostomatology," "Prophylaxis of Dental Caries," and "Periodontosis." Those who wish to collaborate are requested to communicate as soon as possible with I Congreso de Odontoestomatológia, Av. Gral. Rodríguez 8616, Lomas de Zamora, Buenos Aires, Argentina.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

History of Dental Laboratories and Their Contributions to Dentistry. By Robert J. Rothstein. Philadelphia, J. B. Lippincott Company. Price, $7.50.

The Design and Construction of Removable Orthodontic Appliances. By C. Philip Adams. Baltimore. Williams & Wilkins Company. Price, $5.50.

Anomalies of Infants and Children. By D. McCullagh Mayer, New York, McGraw-Hill Book Company, Inc. Price, $12.00.

Clinical Enzymology. By Gustav J. Martin. Boston. Little, Brown & Company. Price, $6.00.